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POLYCHLORINATED BIPHENYL TRANSPORT IN COASTAL MARINE FOODWEBS

by

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CR808006

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U.S. ENVIRONMENTAL PROTECTION AGENCY
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16. ABSTRACT The extent to which polychlorinated biphenyls (PCBs) may be assimilated into fish from dietary sources was studied by providing known doses of PCBs (as Aroclor 1254 in food) to striped bass and analyzing cross-gut transport, tissue distribution and elimination. Assimilation and elimination data from single and multiple doses for whole fish were used to calculate rate-constants for PCB accumulation (k_a) and elimination (k_e) according to one-compartment pharmacokinetic models. The data from analysis of individual tissues were used to calculate k_a and k_e for individual tissue compartments.

The major conclusions from the study are that PCBs in food represent a major source of PCB to fish (up to 80% of total body burdens). The PCBs obtained from food cause a rapid approach to steady state, but are eliminated slowly with a half-time of ~ 120 hr. More than 85% of the PCB ingested with food is assimilated into the tissues. The long-term model showed that PCB burdens in striped bass exposed to food containing different concentrations of PCB will decline slowly when levels in food decline, but increase rapidly (90% plateau reached in 9 doses) when levels in food increase.

Preliminary verification studies support the pharmacokinetic model for PCB accumulation in striped bass with food as the major source.

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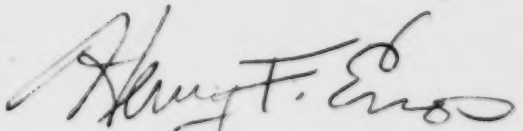
FOREWORD

The protection of our estuarine and coastal areas from damage caused by toxic organic pollutants requires that regulations restricting the introduction of these compounds into the environment be formulated on a sound scientific basis. Accurate information describing dose-response relationships for organisms and ecosystems under varying conditions is required. The Environmental Research Laboratory, Gulf Breeze, contributes to this information through research programs aimed at determining:

- the effects of toxic organic pollutants on individual species and communities of organisms.
- the effects of toxic organics on ecosystems processes and components.
- the significance of chemical carcinogens in the estuarine and marine environments.

PCBs hold a unique position as an environmental contaminant because of their ambient and biological ubiquity. Results reported here have direct practical bearing for assessing the magnitude and extent of Hudson River contamination and for evaluating the incorporation of PCBs into potential human food organisms.



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ABSTRACT

The extent to which polychlorinated biphenyls (PCBs) may be assimilated into fish from dietary sources was studied by providing known doses of PCBs (as Aroclor 1254 in food) to striped bass and analyzing cross-gut transport, tissue distribution and elimination. Assimilation and elimination data from single and multiple doses for whole fish were used to calculate rate-constants for PCB accumulation (k_a) and elimination (k_e) according to one-compartment pharmacokinetic models. The data from analysis of individual tissues were used to calculate k_a and k_e for individual tissue compartments.

The pharmacokinetic data were used to evaluate the importance of PCB uptake from food, estimated body burdens arising from PCB in food, and to calculate a long-term model for PCB accumulation in Hudson River striped bass.

The major conclusions from the study are that PCBs in food represent a major source of PCB to fish (up to 80% of total body burdens). The PCBs obtained from food cause a rapid approach to steady state, but are eliminated slowly with a half-time of ~ 120 hr. More than 85% of the PCB ingested with food is assimilated into the tissues. The long-term model showed that PCB burdens in striped bass exposed to food containing different concentrations of PCB will decline slowly when levels in food decline, but increase rapidly (90% plateau reached in 9 doses) when levels in food increase.

Preliminary verification studies support the pharmacokinetic model for PCB accumulation in striped bass with food as the major source.

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FINAL REPORT
CR 808006

POLYCHLORINATED BIPHENYL TRANSPORT IN COASTAL MARINE FOODWEBS

I. INTRODUCTION

Ecotoxicology of the Polychlorinated Biphenyls: The polychlorinated biphenyls (PCBs) were first used in industry as early as 1929 (Nelson, 1972). Thirty-seven years later Jensen (1966) published the first account of PCBs appearing in the flesh of fishes taken from natural waters. This sudden appearance of PCBs on the environmental scene was misleading, however, in that PCB identification and quantitation were not so much dependent upon their presence as upon the development of suitable analytical techniques for their detection.

Within a few years of the discovery of PCBs in environmental samples, investigators in many locations documented the near-ubiquity of PCB distribution in the global environment, as well as evidence for the toxic effects PCBs may have on organisms (Risebrough et al., 1968). In aquatic systems, the PCBs have been shown to be acutely toxic to shrimp, oysters, Daphnia and various fishes (see reviews in Nelson, 1972; Hutzinger et al., 1974; Wassermann et al., 1979; National Academy of Sciences, 1979). Documented chronic effects among aquatic organisms include decreased rates of growth in oysters, decreased photosynthesis in algae, impaired respiratory function in both vertebrates and invertebrates, altered developmental patterns, skeletal abnormalities and increased susceptibility to disease (Duke et al., 1970; Fisher, 1975; Wildish, 1970; Cantilli, 1979; Mehrle et al., 1982). Even at exposure

levels known not to influence mortality or morbidity directly, PCBs tend to accumulate in aquatic organisms to alarming levels. In the northeastern United States, for example, excessively high levels of PCBs in fisheries products (i.e., concentrations $\geq$ 5.0 µg/g wet weight in edible flesh) have caused closure of commercial fisheries for striped bass (Hudson River), flounder (New Bedford) and lobster (New Bedford). Furthermore, public health advisories recommending reduced consumption of fish due to PCB contamination have been issued in Massachusetts, New York (coastal, estuarine regions and Great Lakes regions) and New Jersey (coastal and estuarine).

Sources of PCBs to the Coastal Marine Environment: The PCBs possess numerous "desirable" industrial characteristics, and were widely used in many industries. A partial list of applications includes: dielectrics, heat exchange fluids, hydraulic fluids, plasticizers, flame-retardant lubricants, pesticide carriers and pigment carriers in printing inks (Nelson, 1972). To a large extent, PCB use in industries was dispersive; little care was taken to prevent wastage or environmental dispersion. Anecdotal information suggests that PCBs have been used as road-sprays to retard dust, and as growth retardants for roadside foliage.

The variety of uses found for PCB resulted in widespread occurrence of PCB in the atmosphere, in surface waters, and in the sediments of lakes, rivers and estuaries. For the most part, the contamination levels seen in such regions can be ascribed to two major sources; industrial discharges and effluent releases from sewage treatment plants (O'Connor et al., 1982; Bopp et al., 1981; NAS, 1979).

In the coastal marine environment there occur rather few direct

industrial discharges; however, discharges from sewage treatment plants are rather common. Thus, for the typical coastline, the major sources of PCB input are likely to be 1) sewage discharge, 2) river runoff, and 3) atmospheric deposition. In the vicinity of major urban-industrial areas, such as Boston, New York, Norfolk, San Francisco and other areas, discharges into estuarine waters from industry and treatment plants increase significantly the quantity of PCB material transported in river discharges. In the New York region the transport of dredged estuarine sediments to ocean dumpsites represents a major source of PCBs to the New York Bight (O'Connor et al., 1982). Likewise, in Long Island Sound, at the Philadelphia Dumpsite and in San Francisco, ocean disposal of dredged material may represent a major source of PCBs to the system, capable of causing significant bioaccumulation in fishes and shellfish (O'Connor and Stanford, 1979; Bopp et al., 1931; O'Connor et al., 1982; O'Connor and Pizza, 1984).

Fate of PCBs in the Coastal Marine Environment: Due to their low solubility and strong adsorptive potential, PCBs in the marine ecosystem are likely to remain associated with finely divided particulates, either in the suspended or deposited state. Because the potential for desorption is low (Di Toro and Horzempa, 1982), release to the water column from either the deposited or suspended state is minimal (O'Connor and Connolly, 1980; Mackay, 1982), and accumulation by biota due to direct water uptake is likely to be low.

In either the geologic or biologic matrix, PCBs are highly persistent; they are neither metabolized nor transformed to any measurable extent by bacteria in the sediment, nor by the mixed function oxidase

system in organisms (World Health Organization, 1976; NAS, 1979; U.S. EPA, 1980). In highly contaminated systems, such as the Hudson River, New Bedford Harbor or the Duwamish Waterway, the persistence of PCB may lead to very high levels of bioaccumulation (see, e.g., N.Y. State Dept. of Environmental Conservation, 1981, 1982; Sloan and Armstrong, 1982; Brown et al. in press, 1985). PCB levels in coastal marine systems, however, are much lower than in estuarine systems, and the levels generally found in marine fishes and shellfish are lower than in estuaries by as much as an order of magnitude (Cahn et al., 1977; Spagnoli and Skinner, 1977; MacLeod et al., 1981; Boehm and Hirtzer, 1982; O'Connor et al., 1982; O'Connor, 1984 in press).

Little is presently known regarding the mechanisms of PCB transport in coastal marine ecosystems. The objective of the Cooperative Research Project between the NYU Medical Center and the EPA Gulf Breeze Environmental Research Laboratory was to investigate and describe PCB transport mechanisms in food webs. This final report describes experiments conducted between October 1980 and October 1983 and provides analyses and interpretation of the results as they pertain to prediction of PCB body burdens in fish populations.

II. OBJECTIVES AND DESIGN

The present Cooperative Research Agreement was part of a larger work unit undertaken by GBERL aimed at describing the extent to which PCBs introduced to the coastal marine environment with dredged material might accumulate in marine fishes. The work unit had two parts. The first, directed by Norm Rubinstein, was carried out at GBERL and was designed to determine from microcosm studies the transport of PCBs from contaminated harbor sediments to fishes, shellfishes and fish food organisms. Results of these studies have been documented (Rubinstein et al., 1983 and in press). The second portion, described in this report, was designed to study the mechanisms of PCB transport between fish food organisms and fishes, and to provide, if possible, a description of the rate constants for PCB assimilation due to dietary uptake. The ultimate objective of the combined studies was to provide a predictive framework for estimating PCB accumulation in marine fishes, and to test the validity of our predictions by comparison of laboratory results with field situations.

Design Development and Chronology: Initial studies encompassing the first portion of Year I (October 1980 through April 1981) attempted to establish the usefulness of techniques, determine proper species for laboratory analysis, and understand potential flaws in the experimental design. These studies are summarized in this section, and relate to overall project execution as "rangefinding" data.

1) Species Selection:

The initial project design called for use of the hogchoker (Trinectes maculatus) and the winter flounder (Pseudopleuronectes americanus) as subjects for research. In fact, several species were used in the rangefinding studies. These included hogchokers and winter flounder, as well as white perch (Morone americana) and striped bass (Morone saxatilis).

Criteria for selection of the major test species were: 1) ecological niche (marine species required); 2) ease of capture and availability; 3) ability of the species to tolerate laboratory holding conditions; and 4) use of the species as a human food resource. In the final analysis, the organism chosen for detailed study was the striped bass. In a subsequent study conducted at Gulf Breeze, we incorporated spot (Leiostomus xanthurus) into experiments, and spot studies were used in a microcosm food-chain experiment (Rubinstein et al., in press).

Preliminary experiments were undertaken, in which we provided fishes with live food introduced directly to the holding aquarium. The food organisms had been previously labeled with ^{14}C-Aroclor 1254 in order to provide a dietary PCB burden directly to the fish. Hogchokers, white perch and striped bass were all treated in this manner. Analyses of ^{14}C-PCB in the holding water and determination of tissue ^{14}C-PCB masses in the fishes after exposure, however, revealed unacceptable differences in the mass balance for ^{14}C-PCB. Most mass balances yielded far more than 100% of the dose administered.

The problems were resolved by administering ^{14}C-PCB to the fish by

gavage, a technique developed in this laboratory (see Pizza and O'Connor, 1983) and applied most successfully to the striped bass. All subsequent food-chain transport studies were performed using the gavage technique, including the studies performed with spot in 1982.

2) Determination of Design:

Successful administration of food by gavage made a food-mediated PCB transport design feasible, providing sufficient care was taken to account in the experimental tanks for the effect on the mass balance of all possible routes of PCB movement. The basic design, therefore, came to be one in which the following elements were considered:

a) biomass of test specimens was maintained at a uniform level

b) known doses of ^{14}C-PCB with a known level of radioactivity were incorporated, such that nanogram levels in specific tissues could be detected

c) excretion of PCB to the water was controlled by exposing fish to the exposure water, but without dietary ^{14}C-PCB exposure

d) ^{14}C-labeling of food was always performed using an ecologically relevant striped bass food item, Gammarus tigrinus (O'Connor, 1984 in press).

3) Other Design Considerations

Two additiional studies were carried out. They were: 1) to determine, under laboratory conditions, the relative importance of dietary and water-derived PCB uptake; and 2) to carry out a field sampling pro-

gram (Year III) aimed at establishing whether PCB transport via the food chain provides a reasonable estimate of body burden in the natural environment.

III. APPLICATION OF THE DATA

The project was designed to provide descriptive data useful in understanding how, and to what extent, PCB transport may occur in coastal marine food-chains. It soon became clear that the data could be applied in a predictive manner if they were interpreted using a thermodynamic, mass-balance approach. Such concepts, first applied to organochlorine pesticides in fishes by Norstrom et al. (1976), were formalized by Thomann (1978, 1981) into a food-chain PCB uptake model. The present study owes much to the concepts developed by Thomann (1981) and Thomann and Connolly (1984), and was partly designed to address questions regarding the proper value for a significant factor in their PCB models; i.e., the "food-chain multiplier".

Since it was possible to monitor administration, assimilation, body distribution and elimination of ^{14}C-PCB in the test species, the results fit precisely the pharmacokinetic models developed by Goldstein et al. (1974) for dose/effect evaluation of pharmaceutical products. By combining the long-term (life-cycle) modeling approach of Thomann (1978, 1981; Thomann and St. John, 1979) with the pharmacokinetic approach, we proceeded to develop a model describing and predicting PCB burdens in a fish as determined exclusively by dietary sources (Pizza and O'Connor, 1983; O'Connor and Pizza, 1984).

It is expected that the data provided herein, as well as the experimental and modeling approach, will be of value to both industry and regulatory agencies that evaluate the long-term ecological consequences of contaminant discharges to the environment.

IV. ORGANIZATION OF THE REPORT

Varied and diverse subjects comprise this report. To provide some coherence, we have organized the report as follows:

Section V describes dietary uptake and pharmacokinetics of PCBs in striped bass. Section VI describes the tissue transport of PCB in striped bass, as well as conclusions regarding routes of PCB transport and elimination. The uptake and elimination data were used in developing a pharmacokinetic model to predict PCB burdens in marine fishes, described in Section VII. Section VIII presents the results of a field study aimed at verifying the pharmacokinetic model for striped bass in the Hudson and coastal Atlantic region. All references are collected in Section IX; the individual sections are organized peer-reviewed publications.

V. DIETARY TRANSPORT OF PCBs IN STRIPED BASS

INTRODUCTION

The entire food web of the Hudson estuary is contaminated with PCB (O'Connor, 1982). It may be assumed, therefore, the PCB accumulation by fishes is due partly to direct uptake from water (i.e., by equilibrium partitioning; see Branson et al., 1975; Clayton et al., 1977; Thomann, 1981; Califano et al., 1982) and partly due to accumulation from the diet (Mayer et al., 1977; Guiney and Peterson, 1980; Bruggeman et al., 1981).

The kinetics of dietary PCB absorption in fishes received little attention for at least two reasons: 1) the difficulty in quantifying secondary uptake of contaminants which desorb or dissolve from food prior to ingestion; and 2) the difficulty in quantifying the ingested dose. These problems were addressed by Guiney and Peterson (1980) in a study where the ingested dose was given to fish (Salmo gairdneri and Perca flavescens) in sealed gelatin capsules. Thus, a known dose was administered to the absorption site without secondary uptake from water, allowing the calculation of precise distribution data and elimination phase kinetics. To our knowledge, alimentary tract absorption rate-constants for PCBs have not been published for any fish species.

This chapter describes experiments in which uniform doses of ^{14}C-Aroclor 1254 were given to striped bass by gavage with live food. Absorption-site and whole-body kinetics related to uptake, elimination and rate of accumulation of PCB body burdens were generated using the principles of drug accumulation (Goldstein et al., 1974).

MATERIALS AND METHODS

Young-of-year striped bass were collected from the Hudson River at Stony Point, N.Y. The fish were maintained at 2 parts per thousand (o/oo) salinity at ~ 20°C in activated-carbon filtered aquaria for 7-14 days prior to experiments. Food was minced earthworms, Daphnia spp., and Gammarus tigrinus; no food was given for 24 hr prior to dosing. The weight of fish used in the single-dose study was 0.88 ± 0.04 g (dry wt; $\bar{x} \pm s_{\bar{x}}$). Fish used in the multiple-dose study weighed 0.75 ± 0.04 g (dry wt).

Gammarus tigrinus, an estuarine amphipod which occurs frequently in the diet of Hudson River striped bass, was used as the food organism. G. tigrinus were cultured in the laboratory by the procedures of Ginn (1977). Groups of 300 mature individuals (~7 mm total length) were labeled with PCB by exposure for 24 hr to 10 µg/L ^{14}C-PCB (uniformly ring-labeled Aroclor 1254; New England Nuclear) at 2 o/oo salinity (Peters and O'Connor, 1982). The animals were then removed from the exposure chamber, rinsed in ^{14}C-PCB-free water, and blotted to remove excess water. Labeled G. tigrinus were then loaded into glass tubes (I.D. = 3 mm) to a set quantity, determined from preliminary studies as that amount per feeding (18 mg, dry wt) which filled a striped bass stomach without overextension or forcing food into the intestine.

Food given by gavage had a nominal dose of 500 ng PCB per feeding. Feedings were performed by inserting the loaded glass tube into the esophagus; the ration was gently extruded into the stomach by the action of a plunger in the tube. The fish were then transferred to activated-carbon filtered aquaria at the holding conditions. At the time of each

feeding, some food tubes were injected into scintillation vials for estimation of dose.

Secondary uptake was measured directly during a multiple-dose "barrier" study. Aquaria were divided into two parts by barriers which allowed free exchange of water, but prevented passage of fish. In the experiment conducted, the fish for estimate of secondary uptake received two sham feedings of G. tigrinus, and were held isolated from, but in the same tanks with, equal numbers of fish which had received two feedings of ^{14}C-PCB. Since the only source of ^{14}C-PCB in the tanks was that given the experimental fish by gavage, any ^{14}C-PCB in the control fish at the end of the exposure was the result of water uptake of PCB eliminated from ^{14}C-PCB labeled fish.

Single-dose and multiple-dose experiments were conducted. In the single-dose study, fish were force-fed and sampled at 6, 12, 24, 48, 72, 96 and 120 hr after feeding. Fish in the groups held for 96 and 120 hr were fed live ^{14}C-PCB free G. tigrinus during the holding period.

In the multiple-dose study, fish were force-fed 387 ± 13 ng ^{14}C-PCB/dose ($\bar{x} \pm s_{\bar{x}}$; n = 14) with dosing interval of 48 hr. Subgroups were analyzed at the end of each interval after receiving 1, 2, or 3 doses.

Care was taken to reduce secondary uptake of excreted PCB by removing feces, and continuous filtration of holding water through an activated charcoal system.

Fish were stunned by immersion in ice-water and dissected, with organs and remaining carcass sections placed in individual glass scintillation vials with teflon caps. The samples were dried to a constant

weight at 50°C. After weighing, the samples were wetted with deionized water and solubilized in Protosol (NEN) for 24 hr at 50-55°C. Where necessary, samples were decolorized with 30% hydrogen peroxide at 50°C for 30 min. After cooling, 18 mL Econofluor (NEN) cocktail was added. Samples were counted on a Packard Tri-Carb Model 460 CD liquid scintillation counter. Background radiation and sample quench were accounted for, and the instrument was run in the external standard, automatic efficiency control mode.

Alimentary tract and whole-body levels of contaminant were determined for subsequent rate-constant calculations. Organ-specific levels were also determined, and will appear in a later publication.

RESULTS AND INTERPRETATION

Secondary absorption of excreted ^{14}C-PCB was minimized during the current work. The whole-body levels in both the ^{14}C-PCB and sham exposed fish of the barrier study were determined in order to calculate percent dietary uptake. After administration of two radiolabeled feedings (cumulative dose: 980 ng), diet was responsible for 98.3% of the body burden.

Data from the single-feeding studies were normalized to percent dose administered. The "fraction absorbed per unit time" should be independent of actual dose (Bederka et al., 1971; Goldstein et al., 1974). We tested this by comparing percent absorption of two ^{14}C-PCB doses differing by a factor of 2.5 (380 ng vs. 965 ng); the fractions absorbed at 48 hr were not significantly different (Student's Test of t; $P > 0.05$).

The amount of PCB remaining at the site of absorption (alimentary tract) decreased over time with two obvious phases (Table I, Figure 1). A phase of rapid decrease occurred between 0 and 24 hr, defined by a regression line with slope = -0.0448. The line defining alimentary tract PCB decrease between 48 and 120 hr had a slope = -0.0021. Since whole-body PCB burden was > 80% of dose after 48 hr, and the alimentary tract lost PCB rapidly (< 4% of dose remaining after 48 hr) (cf. Figures 1 and 2), we concluded that the initial, rapid loss from the alimentary tract represented transport of PCB from the gut to the remainder of the body. The slower removal after 24 hr was similar to the whole-body elimination rate, and represented tissue elimination rather than absorption. It should be noted that PCB eliminated with the feces by 24 hr, as monitored in several experiments, accounted for < 10% of the administered dose. In general, the alimentary tracts of the striped bass were not cleared of food until about 24 hr after feeding. Thus, for the purpose of estimating kinetic constants, we assume that the full dose was present in the fish during the period of rapid removal from the alimentary tract.

PCB elimination from the body began soon after oral administration (Table II, Figure 2). The data show first order elimination over time, the regression yielding a slope of -0.0023.

The results of the multiple feeding study show that for separate doses of 387 ± 13 ng ^{14}C-PCB with a dosing interval of 48 hr, a total of 684 ± 36 ng or 58.9 percent of the cumulative dose (1161 ng) was retained at the end of the third interval (Table III).

These data may be applied to kinetic models describing the

TABLE I. Absorption of PCB from single dietary exposure. The reduction of alimentary tract PCB level with time as a percentage of the administered dose.

Time (hr)	Percent Unabsorbed Dose $\bar{x} \pm s_{\bar{x}}$	n
0	100.00 ± 1.89	10
6	76.54 ± 2.11	10
12	44.70 ± 5.49	10
24	7.03 ± 0.39	3
48	4.08 ± 0.26	5
72	3.38 ± 0.48	5
96	3.54 ± 0.20	10
120	2.87 ± 0.31	5

Figure 1. Percent unabsorbed dose as a function of time. PCB removal from the alimentary tract as determined by two processes: 1) absorption of administered dose (phase 1); and 2) elimination from tract tissue (phase 2).

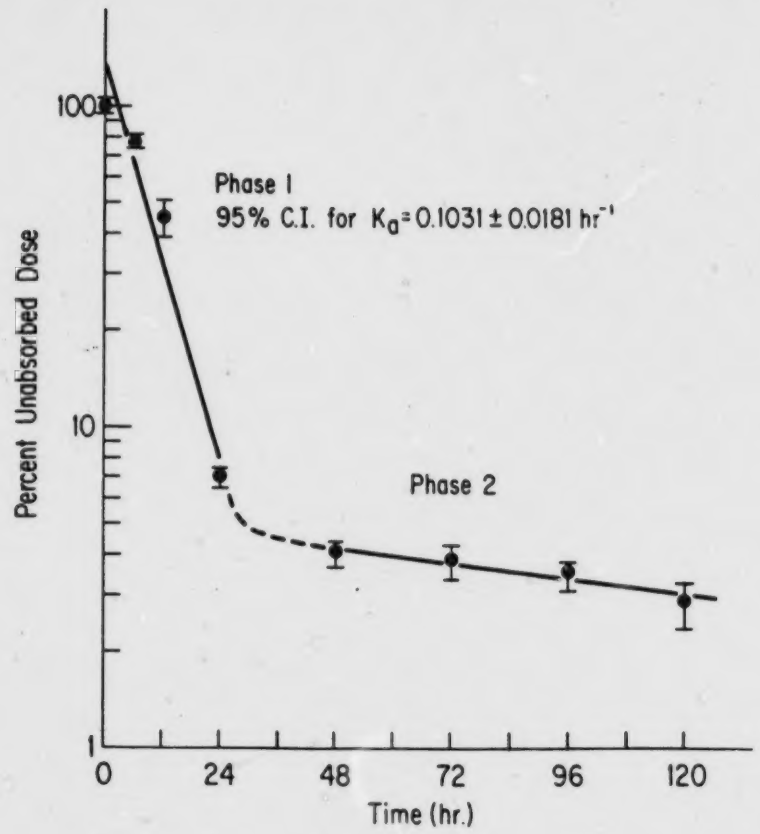

Percent Unabsorbed Dose
100
10
1
Phase 1
95% C.I. for $K_a = 0.1031 \pm 0.0181\ hr^{-1}$
Phase 2
0
24
48
72
96
120
Time (hr.)

TABLE II. Whole-body elimination of PCB after single dietary exposure. The reduction of body PCB level with time as a percentage of administered dose.

Time (hr)	Percent Dose in body $\bar{x} \pm s_{\bar{x}}$	n
0	100.00 ± 1.89	10
6	93.21 ± 2.37	10
12	83.73 ± 2.29	10
24	87.33 ± 1.27	3
48	77.65 ± 6.38	5
72	66.85 ± 5.46	5
96	60.51 ± 2.35	10
120	46.99 ± 4.29	5

Figure 2. Percent dose in body as a function of time. PCB elimination from whole-body after a single dietary exposure.

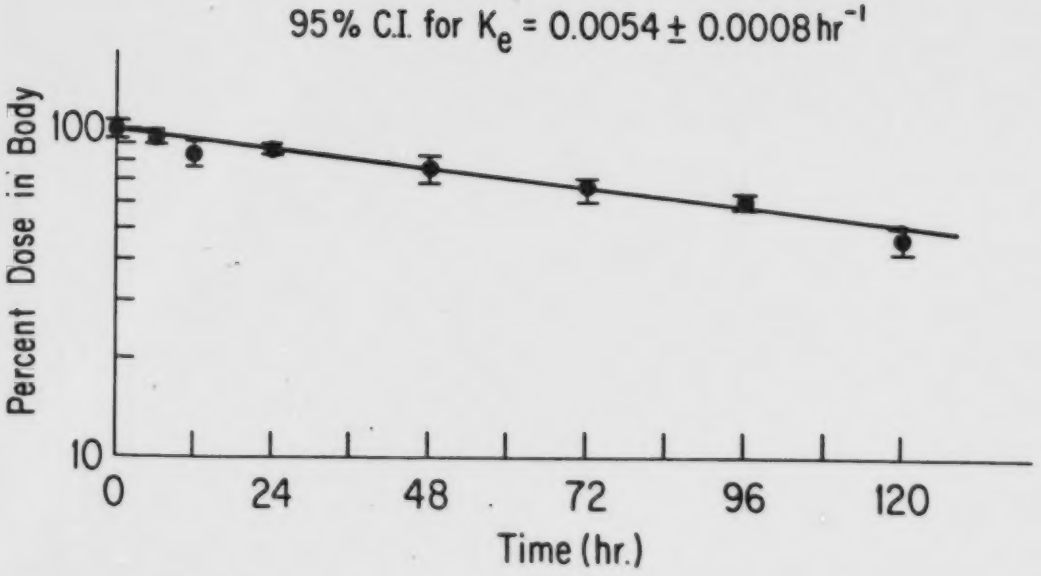

95% C.I. for K_e = 0.0054 ± 0.0008 hr^{-1}
Percent Dose in Body
100
10
0
24
48
72
96
120
Time (hr.)

TABLE III. Cumulative whole-body PCB level from multiple dietary exposure.

Doses Given	Cumulative Dose (ng)	PCB Retained at End of Interval ($\bar{x} \pm s_{\bar{x}}$)
One	387	295.0 ± 24.2
Two	774	504.9 ± 53.4
Three	1161	683.9 ± 38.1

incorporation of chemical compounds into organisms which function as single compartments. The equations and notation are from Goldstein et al. (1974). The kinetic model uses common mass balance equations; we have minimized examples of derivation or rearrangement, therefore, and present only those expressions which apply to the interpretation of our experimental results.

The first-order kinetics of dietary uptake of PCB can be described by following the time-course of elimination of the compound from the absorption site (alimentary tract). The equation defining this process is:

$$M = M_o e^{-k_a t} \qquad (1a)$$

$$\log M = \log M_o - k_a t/2.30 \qquad (1b)$$

where M_o is the quantity of compound placed at the absorption site at time 0, M is the quantity remaining at time t, and k_a is the absorption rate-constant. The absorption rate-constant may be obtained by regression of the log unabsorbed dose against time, since Equation 1b represents a straight line with a negative slope = $k_a/2.30$. The k_a may also be determined directly by exponential curve fitting. Applying regression analysis to the data from the absorption phase (0-24 hr of gut clearance; Table I, Figure 1) yields a 95% confidence interval (C.I.) for the k_a equal to 0.1031 ± 0.0181 hr^{-1}.

The absorption <u>half-time</u> for PCB from the gut can be determined by:

$$\text{Absorption } \underline{\text{half-time}} = (-\ln 0.5)/k_a \qquad (2)$$

The reader should note that in this discussion, the terms <u>half-time</u> and <u>half-life</u> are used. By convention, each has the notation $t_{1/2}$. To

avoid confusion, we have chosen to delete notation and refer to half-time specifically as relating to the process of PCB absorption or shift in "steady state". Half-life will be used only in relation to the elimination of PCB from the body.

Striped bass given a single dose of PCB with $k_a = 0.1031\ hr^{-1}$ have a calculated absorption half-time (the time for the PCB source to be depleted by 50 percent) of 6.7 hr. The sharp change in slope after 24 hr (3-4 half-times) represents the end of PCB assimilation from the gut, and coincides with the time of food clearance from the alimentary tract. After 24 hr, PCB depletion from the gut enters a second phase which reflects tissue elimination of PCB rather than absorption into the body (see below).

The equation for calculating the rate of elimination of PCBs from the body is the same as that used for absorption rate-constants, but with different notation:

$$X = X_o e^{-k_e t} \qquad (3a)$$

$$\log X = \log X_o - k_e t/2.30 \qquad (3b)$$

where X_o is the quantity of compound present in the body at time 0, X is the quantity present at time t, and k_e is the elimination rate-constant. The k_e was determined from the slope of the regression line (as in Figure 2) from the whole-body data (Table II). The half-life for elimination is determined from k_e using Equation 2 with appropriate changes in notation.

The 95% C.I. of the whole-body elimination rate-constant for PCB in striped bass was $0.0054 \pm 0.0008\ hr^{-1}$, equal to an elimination half-life

of 128 hr. Whole-body elimination exhibits a single phase from the time of administration to termination at 120 hr. Alimentary tract elimination of PCB (see Figure 1; phase 2 of absorption) had $k_e = 0.0047 \pm 0.0037\ hr^{-1}$ (C.I.), a value similar to the rate-constant for whole-body elimination. The corresponding half-life was 146 hr. This confirms that the alimentary tract and whole-body were similar with respect to PCB elimination.

Rate-constants for PCB elimination from the whole-body and the gut were small ($\leq 5\%$) relative to PCB absorption (k_a) of the initial, rapid phase. We chose not to apply a k_e correction factor to the k_a used in our calculations for two reasons. First, some of the PCB elimination from the whole-body is via the hepatic pathway (Pizza, unpublished data). Biliary excretion of contaminants, such as PCB, makes them re-available for assimilation in the gut. This input to the alimentary tract during the absorption phase would result in a measure of k_a lower than the actual value.

Second, the data used in the kinetic model for absorption and elimination (Tables I and II) show deviation equal to or greater than the 5% by which we might correct the k_a value by subtracting the k_e. Rather than yielding a more meaningful k_a, the correction would compound the experimental error without adding to or improving the reliability of the outcome of the pharmacokinetic model we present. For these reasons, we assume biliary input and tissue output to offset one another, and we ignore both in our determination of absorption rate.

The k_a and k_e derived for striped bass can be used to determine percent absorption of single PCB doses and provide insight into the

temporal relationships between body burden and absorption rate. The equation used to calculate the fractional absorption of a given dose over time is:

$$X/H_o = 1/[(k_e/k_a)-1]\ [e^{-k_e t/(k_e/k_a)} - e^{-k_e t}] \qquad (4)$$

where X/H_o is the amount of PCB in the body (X) relative to the dose (H_o) placed at the site of absorption at time 0. We showed earlier that the fractional absorption of PCB in striped bass was independent of the actual dose.

For any dose, the time at which the maximum whole-body dose is accumulated (t_{max}) is calculated for the situation where $k_a \neq k_e$ by:

$$t_{max} = 2.3/(k_a-k_e) \log k_a/k_e \qquad (5)$$

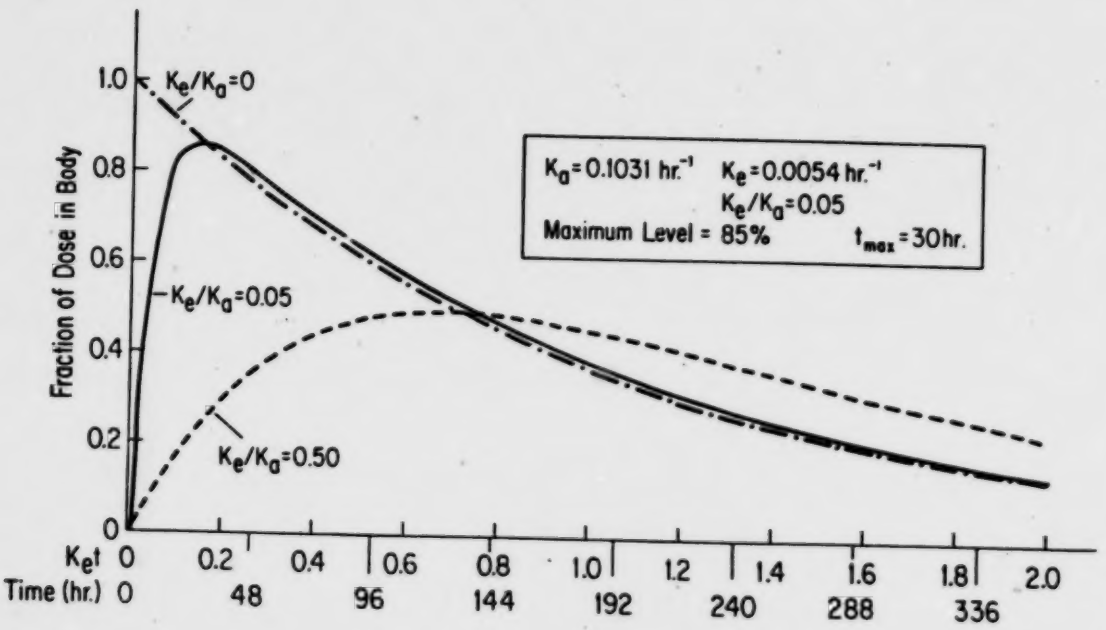
and the maximum fraction attained from a single dose (X_{max}/H_o) where $k_a \neq k_e$ can be found by:

$$X_{max}/H_o = (k_a/k_e)^{k_e/(k_e-k_a)} \qquad (6)$$

The single-dose data from this study (Tables I and II) yielded a maximum PCB level (X_{max}/H_o) equal to 85% of the dose, at 30 hr after application of the dose (t_{max}). A graphical presentation of fractional absorption of PCB in striped bass as determined from the k_a and k_e of our single-dose study is given in Figure 3. The units of the X-axis are presented as $k_e t$ in order to normalize for the elimination rate-constant. The units can be converted to actual time by dividing by k_e.

The absorption rate of the single dose study was rapid, and it should be noted that the approach taken yielded a conservative estimate of k_a. Had the PCB dose been administered in a more bioavailable matrix

Figure 3. The fractional retention of a single dose by first-order absorption and elimination. The solid curve was determined from Equation 4 and the data of the current study where $k_e/k_a = 0.05$. A maximum of 85 percent was absorbed at 30 hr. The other curves are presented for comparison (Goldstein et al., 1974) at the same k_e. The case where $k_e/k_a = 0$ is attained by constant input. For $k_e/k_a = 0.50$, a lower maximum would be attained at a later time.


1.0
0.8
0.6
0.4
0.2
0
Fraction of Dose in Body
K_e/K_a=0
K_e/K_a=0.05
K_e/K_a=0.50
K_a=0.1031 hr.$^{-1}$ K_e=0.0054 hr.$^{-1}$
K_e/K_a=0.05
Maximum Level = 85% t_{max}=30hr.
K_et 0 0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8 2.0
Time (hr.) 0 48 96 144 192 240 288 336

(e.g., emulsified lipid in the intestine), rather than in a live food organism which required digestion, this study may have yielded a more meaningful and larger k_a. Alimentary tract PCB absorption has been shown to increase by improved bioavailability (R.D. Vetter, Univ. of Georgia, Athens GA, pers. comm.; J.M. O'Connor et al., unpublished data).

Accumulation of PCB from constant input (multiple exposure) can be predicted from single-dose studies (Morales et al., 1979). When multiple doses are given, and the concentration of PCB in the fish (C_f) is at steady state, the input and output rates are equal. The body burden (µg PCB/g body wt) can be determined by dividing the input rate (µg PCB/g body wt/hr) by k_e (hr^{-1}). This should not be confused with the terminology presented by Branson et al. (1975) where for equilibrium from water exposure, C_f equaled the water concentration (C_w) times the uptake rate-constant (k_1) divided by the elimination rate-constant (k_2). In that work, the bioconcentration factor was given as k_1/k_2.

For the current work, the bioaccumulation factor (BAF) is not given by k_a/k_e. For a given PCB concentration in the prey (C_p), the bioaccumulation factor at steady state is equal to the feeding rate (g prey/g fish/hr) times the PCB absorption efficiency divided by k_e (Brugseman et al., 1981). The absorption efficiency from the single dose study was ~ 85%. A daily ration of 10% body weight and 85% efficiency would yield a BAF of 0.66. Whether this BAF is realistic can only be determined after a thorough examination of "steady state".

The interpretive approach taken for multiple-dose studies assumes that a compound can be administered at a constant rate (zero-order kinetics) in a regimen where a dose (M_o) is given at regular intervals

(t*). Note that these conditions are met for organisms in nature by feeding at regular intervals on a ration which is determined by metabolic requirements of the animal. Zero-order in this case refers to a constant rate of absorption which is independent of the unabsorbed quantity. It ignores the fluctuations shown to exist from first-order kinetics of single-dose studies.

Such work was done by McLeese et al. (1980), where lobsters (Homarus americanus) were fed PCB-contaminated mussels (Mytilus edulis) every 48 hr for 6 wk. With $k_e = 0.04\ wk^{-1}$, 90% of the plateau would be reached in ~ 60 weeks. The constant input rate can perhaps most easily be maintained during continuous-flow uptake studies with constant contaminant levels. Branson et al. (1975) have shown this in the "accelerated test", where for $k_e = 0.21\ wk^{-1}$, 99% of plateau would be achieved after 22 weeks of continuous exposure to 2,2',4,4'-tetrachlorobiphenyl.

Immediately following administration of a PCB dose, M_o, at t = 0, the level (X) in the body equals M_o. At the end of the dosing interval, where t = t*, the amount in the body is given by Equation 3a, with t = t*.

At the end of any given dosing interval, and just prior to administration of the next dose, the level of PCB in a striped bass is given by the expansion:

$$X = X_o(e^{-k_e t^*})^1 + X_o(e^{-k_e t^*})^2 + \ldots + X_o(e^{-k_e t^*})^n \qquad (7)$$

where the exponent 2...n represents the number of the dose in the series.

The k_e's calculated from the striped bass data (Table III) for each

interval of the multiple dose study were in good agreement (0.0059 ± 0.0001 hr^{-1}, $\bar{x} \pm s_{\bar{x}}$); and agreed also with the single-dose study (k_e = 0.0054 ± 0.0008 hr^{-1}. Our k_e for striped bass of approximately 1 g (dry wt) was determined over a period of one <u>half-life</u>. Generally, elimination is observed for longer periods of time. We chose to limit elimination to 5-6 days in order to avoid the confounding effects of radiotracer dilution which result from growth (Guiney et al., 1977). The discussion of multiple PCB exposure in striped bass will deal with the k_e (0.0056 hr^{-1}) calculated for the cumulative data applied to Equation 7 with the exponent n = 3.

Just <u>after</u> the nth dose is administered, the accumulated body burden (X_n) is:

$$X_n = X_o\,(1-e^{-k_e t^* n})/(1-e^{-k_e t^*}) \qquad (8)$$

and as n becomes large, a plateau level (X_∞) is approached, defined by:

$$X_\infty = X_o/(1-e^{-k_e t^*}) \qquad (9)$$

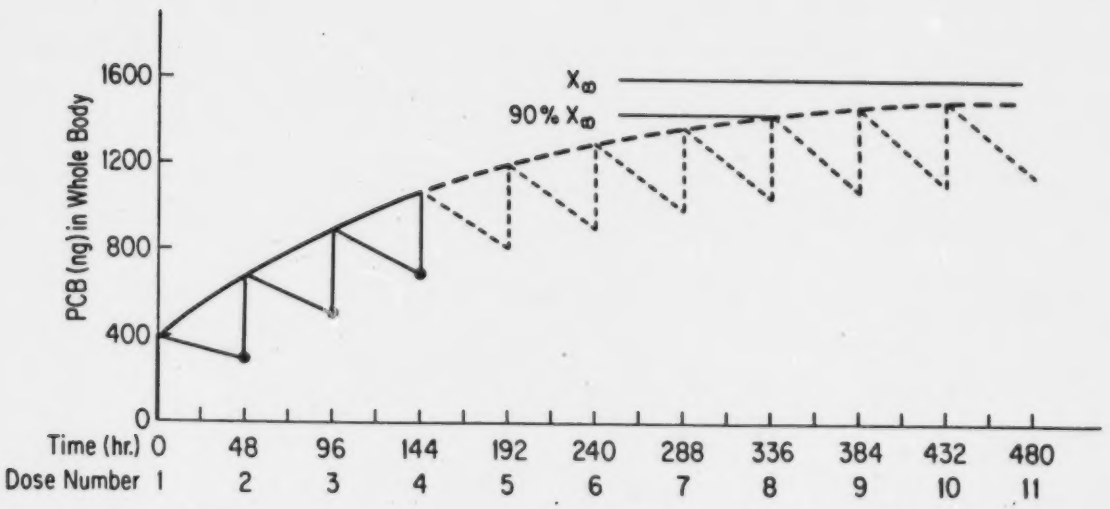
The fraction of plateau (f) attained by a certain number of doses is given by:

$$f = 1-e^{-k_e t^* n} \qquad (10)$$

The number of doses (n) needed to attain a certain fraction of plateau can be determined by rearrangement of Equation 10.

The plateau level for the present study was calculated to be 1592 ng PCB for individual doses of 387 ng PCB given at t* = 48 hr (Figure 4). Under the conditions of our experiment, plateau (99%) would be reached by dose number 17 (total time 32 days). An actual level of

Figure 4. Curve for the cumulative retention of PCB from multiple dosing. Solid lines present the actual levels attained during the experiment. Dashed lines are the calculated extension of the data. The plateau burden (X_∞) is the steady state level attained from peak values after sufficient dosing (see text).


PCB (ng) in Whole Body
1600
1200
800
400
0
X_∞
90% X_∞
Time (hr.) 0 48 96 144 192 240 288 336 384 432 480
Dose Number 1 2 3 4 5 6 7 8 9 10 11

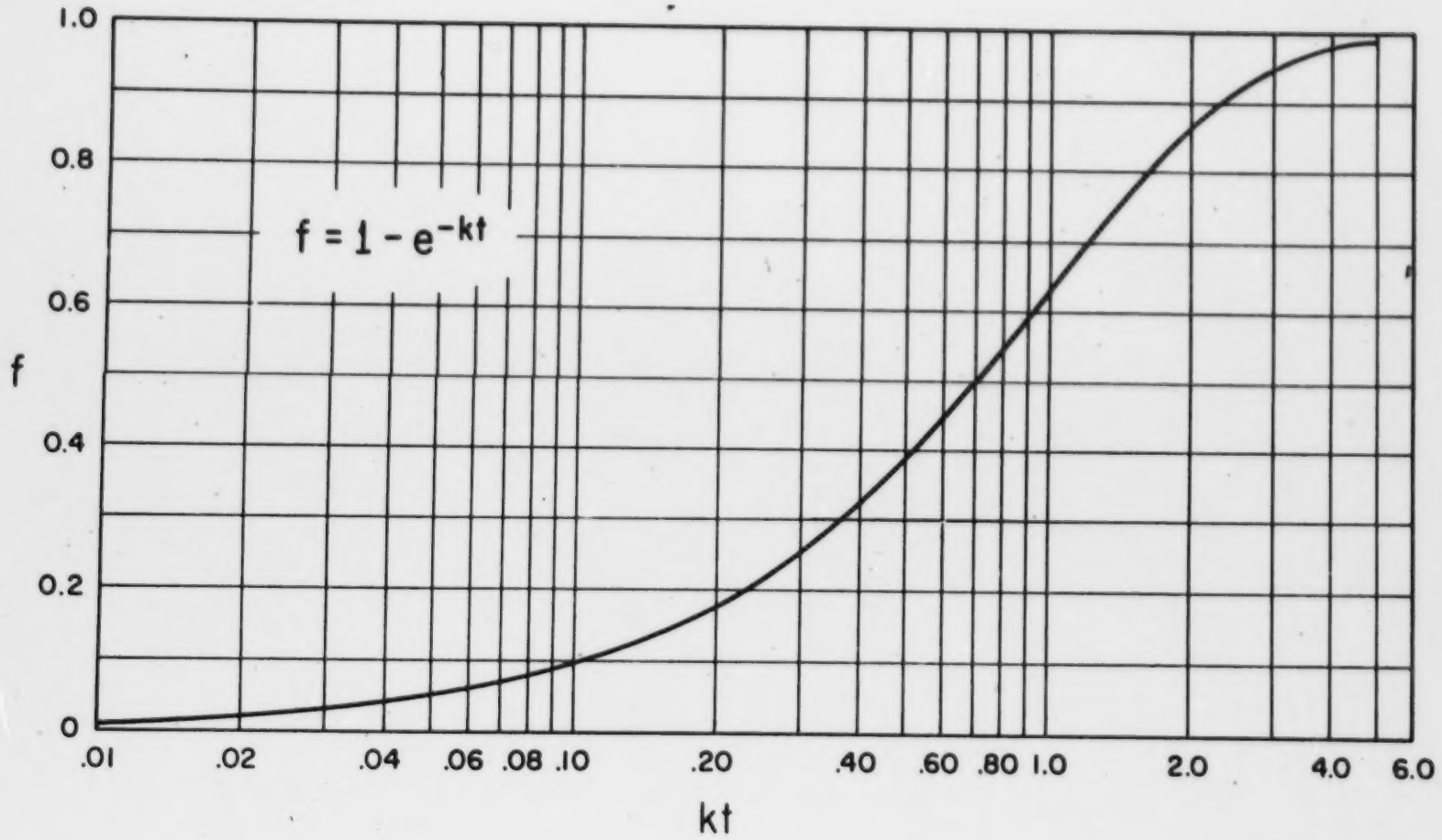
67.2% of the calculated plateau was reached after the fourth dose; 90% of plateau would be attained at 8.3 doses.

Because PCB elimination occurs between doses (k_e = 0.0056 hr^{-1}) the body burden will fluctuate while increasing. At plateau (X_∞) the fluctuation will be equal to X_o/X_∞; the burden returning to a constant level (X_∞) after each dose. We calculated that, in striped bass, the plateau level should fluctuate by 24.3% between doses, if the doses were given at 48 hr intervals (see Figure 4).

A smaller dose and shorter dosing interval would reduce the fluctuation in body burden. In these experiments, we administered an amount of PCB which, in the field, could realistically be ingested over a 48-hr period. PCB concentrations in Gammarus spp. from the brackish water portion of the Hudson River were between 2 and 10 µg/g (dry wt) during the period 1978 to 1981 (O'Connor, 1982). A growth ration of 10% body weight per day for striped bass of ~ 0.8 g dry wt amounts to a PCB ingestion rate of from 160 to 800 ng per day, or 320 to 1600 ng per 48-hr interval. To administer a smaller dose at a proportionately shorter interval would have resulted in less fluctuation, but such a regimen would have been technically unfeasible. It would also be unnecessary, since for a known k_e, the relation X_∞/X_o can be determined for any dosing interval.

Note that when total time (t = t*n) is used in Equation 10, we define the rate of shift from one "steady state" to the next. By solving the equation for f = 0.5, the half-time of the shift is shown to be equal to the half-life for elimination. The value f as a function of time can be obtained from Figure 5 (from Goldstein et al., 1974) for any

Figure 5. The fractional shift to steady state. For a system with constant input rate and first-order elimination, k_e determines the time required to attain a certain fraction (f) of plateau. (With permission from Goldstein et al., 1974.)


1.0
0.8
0.6
0.4
0.2
0
f
$f = 1 - e^{-kt}$
.01
.02
.04
.06
.08
.10
.20
.40
.60
.80
1.0
2.0
4.0
6.0
kt

system with zero-order input, first-order output, and known k_e.

With a half-life of 128 hr, changes of PCB input rate or output rate-constant would cause a very slow shift to a new steady state. Thus, the assumption of steady state being achieved as time approaches infinity may be unfounded in any system other than the laboratory. Annual cycles of growth, feeding, reproduction and migration of fish impose a situation where the organism is, perhaps, always in the process of shifting steady state. Physiological changes which occur over discrete seasons of growth are certain to cause shifts from one steady state to the next (Thomann, 1981). Further changes due to seasonably variable behavior would perpetuate the change (see, e.g., data of Weininger, 1978). More data are needed on the variability of PCB elimination constants in fishes, especially the influence of organism size, growth and physiological condition on k_e.

The relationship of plateau to dose directs attention to some of the key factors in influencing bioaccumulation. The plateau level (X_∞) is dependent on PCB dose (X_o), elimination rate-constant (k_e), and the dosing interval (Equation 9). When k_e and dosing interval (t^*) are relatively constant, as they are for an organism at a given time in its life-history, the absolute quantity of contaminant ingested at each meal is the determinant of the body burden. The quantity ingested (X_o) depends on the contaminant level in the food (C_p) and the biomass of food (ration) ingested during each interval.

This case is an idealized situation for a striped bass of fixed weight and metabolic rate, feeding on a set maintenance ration. We can only determine short-term increases in body burden under such

conditions. To predict bioaccumulation, the experimental design and subsequent modeling attempts must consider growth and the resulting decline in elimination rate (Norstrom et al., 1976; Thomann, 1981).

One might expect growth to reduce k_e, since the metabolic rate of striped bass declines with increased weight (Neumann et al., 1981). Norstrom et al. (1976) indicated that body weight affected clearance rate, but not necessarily via metabolic rate. Calirano et al. (1982) observed a decrease in k_e between larval and young-of-year striped bass exposed to PCBs in water. Such a reduction in k_e dictates a body burden greater than that derived from BAF calculations which do not consider the effects of growth.

The k_e from our single-dose study was applied to a BAF calculation where PCB input rate was constant and elimination was first-order. In this calculation, a young-of-year striped bass was assumed to feed twice a day, ingesting a daily ration of 10% body weight. The hypothetical fish grew over a 5-month period to 1.6 g (dry), and as an approximation of the reduction in elimination rate, the k_e declined with growth as determined by weight to the -0.3 power. The k_e declined to 0.0044 hr^{-1} and the computed BAF was 0.76. This value is much lower than the BAF = 0.95 which would be calculated by simply dividing the feeding rate by the k_e determined after 5 months of growth (0.0044 hr^{-1}). The discrepancy is explained by the fact that the BAF = 0.95 would be attained only with sufficient time (~ 44 days) and cessation of the decline in k_e, allowing for the establishment of a new steady state. This underscores the importance of recognizing that bioaccumulation is rarely characterized by steady state, but rather is a process of uninterrupted plateau shift operating as a continuum.

Data are available for young-of-year striped bass taken in 1978 from the Indian Point portion of the Hudson River (Califano et al., 1982; Mehrle et al., 1982). Concentrations of PCB in these two samples were 1.59 and 2.62 µg/g (wet wt), or about 6.4 and 10.4 µg/g (dry wt), respectively. O'Connor's (1982) data for striped bass food organisms (Gammarus spp.) from the same portion of the Hudson averaged ~ 7 µg/g (dry wt) PCB. Given a BAF of 0.76 and a food source with 7 µg/g PCBs, one might expect a fish to contain 5.3 µg/g PCB (dry wt) due to diet alone.

This calculation suggests that 51 to 83% of the PCB in striped bass is due to dietary uptake. We do not imply that this might be the case for fishes in general; direct uptake from water is an important source of PCB to fishes, and cannot be ignored. However, the exercise does suggest that the dietary PCB component may be of great significance to fishes, especially in heavily contaminated environments such as the Hudson estuary.

Dietary accumulation is most strongly influenced by feeding and clearance rates. The decline of these rates as growth occurs will determine the ultimate dietary contribution to body burden in mature fish. Certain environmental factors, such as reduced temperature, could serve to decrease k_e and increase "BAF" due to a depressive effect on metabolic rates in striped bass and other fishes (Neumann et al., 1961). The impact of the reduced k_e under low temperature conditions, however, might be offset to some extent by simultaneous reduction in feeding during the winter months, or by metabolic compensation to slowly changing environmental conditions (Fry, 1971; Vetter, 1982).

The concept of growth-related plateau shift has application to estimating body burdens in fishes exposed to varying conditions of PCB input during different life-history stages. We know, for example, that when Hudson River striped bass migrate from riverine nursery areas to the lower estuary and marine waters, they show a significant reduction in PCB body burden (LMS, 1980; MacLeod et al., 1981; O'Connor et al., 1982). This reduction should be due to reduced PCB input, since the PCB in both water and food in coastal regions is less than in the estuary (Pierce et al., 1981; O'Connor et al., 1982). With knowledge of the operant k_e and the PCB levels in food and water, the body burden and time for reaching the new and lower level should be calculable, and will be treated in a later chapter.

VI. TISSUE DISTRIBUTION OF PCB AND ROUTES FOR ELIMINATION

INTRODUCTION

The contamination of the Hudson River and estuary with polychlorinated biphenyls (PCB) has been under study for more than ten years (Carcitch and Tofflemire, 1982). Since the first published reports of PCB in Hudson River fishes (Nadeau and Davis, 1976), a great deal of research has centered on describing the physical transport of PCBs in the Hudson system, and estimating trends in PCB body burdens for important fisheries products, such as American shad (Alosa sapidissima) and striped bass (Morone saxatilis) (Turk, 1980; Turk and Troutman, 1981; Armstrong and Sloan, 1980; Pastel et al., 1980; Sloan and Armstrong, 1982).

Several investigators have shown that different fish tissues accumulate PCBs to varying degrees. Guiney et al. (1977) and Narbonne (1979) showed that the liver concentrated PCBs to greater levels than other tissues in yellow perch, rainbow trout and some estuarine fish species. Califano (1981) showed that the liver had the greatest rate of PCB accumulation in tissues of striped bass; the fractional distribution of the whole-body PCB burden was proportional to estimates of blood supply to different tissues. In general, the distribution of PCB in fish tissues has been linked to lipid concentrations in tissues (Lieb et al., 1974; Guiney and Peterson, 1980; Bruggeman et al., 1981).

In this section, we report studies carried out to determine PCB accumulation potential in tissues of striped bass, as well as estimates of PCB elimination rate-constants from different tissue compartments.

MATERIALS AND METHODS

The data for determining PCB accumulation and elimination from striped bass tissues come from the same experiments used in our study of whole-body PCB uptake from dietary sources (Section V; Pizza and O'Connor, 1983). Detailed methods for dosing striped bass with known quantities of ^{14}C-labelled Aroclor 1254 (New England Nuclear Corp.; NEN) are given in that section.

All the striped bass used in these studies were taken from the Hudson River at either Stony Point or Croton Point, transported to the laboratory, and held for a minimum of one week prior to use in experiments. All dosing with ^{14}C-PCB was done by gavage, using Gammarus tigrinus which had previously been allowed to accumulate known doses of ^{14}C-PCB (Peters and O'Connor, 1982; Pizza and O'Connor, 1983). Quantities of ^{14}C-PCB in all the tissue and feces samples taken for analysis were determined by liquid scintillation counting (LSC) on a Packard Tri-Carb LSC. Samples were dried to a constant weight at 50C, weighed, wetted with 0.1 mL deionized water and solubilized in 1 mL Protosol (NEN), decolorized (if necessary) with 0.1 mL 30% H_2O_2 and fluored with Econofluor cocktail (NEN). Concentrations of ^{14}C-PCB in water were determined by LSC after extracting water samples on a Waters C-18 Sep-Pak cartridge (Pizza, 1983).

Three experiments were conducted in order to assess PCB distribution and elimination from tissues and organs. These were: 1) a single dose study to assess short-term PCB uptake and tissue distribution as well as subsequent elimination; 2) a multiple dose (n = 3) study per-

formed to compare tissue burdens, proportional distribution and elimination of larger PCB doses; and 3) a secondary uptake study carried out as a control to determine the percent nondietary uptake of PCB, as well as tissue distributions.

In the single dose study, young striped bass (0.88 ± 0.04 g dry weight) received a single dose of ^{14}C-PCB in Gammarus; subsamples of fish were taken 6, 12, 24, 48, 72, 96 and 120 hr after dosing. Levels of ^{14}C-PCB were determined for gill, liver, gall bladder, alimentary tract, brain, head and remaining carcass. PCB levels in fecal matter were measured at 24, 48, 72 and 96 hr. PCB levels in the water were determined at 6, 24, 72 and 96 hr.

Young-of-year striped bass weighing 0.78 ± 0.04 g (dry wt.) were used in the multiple dose study. Prior to each feeding, a group of G. tigrinus were radiolabeled for 24 hr. The fish received 3 radiolabeled feedings with a dosing interval of 48 hr.

The fish were held in aquaria under the conditions of the single-dose study. Forty-eight hours after the first radiolabeled doses were administered, a subgroup of fish was taken for analysis. At this time, the remaining fish received a second dose. Another subgroup was taken for analysis 48 hr after this second feeding, and the remaining fish were given the third and last dose. Groups of fish were analyzed thereby at the end of each interval after receiving 1, 2, or 3 doses. Gill, liver, gall bladder, alimentary tract, spleen, heart, head, carcass, and a section of epaxial muscle were analyzed by LSC. Feces ^{14}C-PCB concentrations were monitored at 24 hr intervals. The PCB concentration in water was determined at 24, 72, and 120 hr.

The secondary absorption study was performed to determine the extent to which PCBs excreted to the water after accumulation from dietary sources were re-absorbed by the experimental fish. A glass barrier was fitted to the center of each tank. The barrier permitted passage of water across the top two-thirds of the tank; the bottom third was fitted with a solid glass sheet. Four such barrier tanks were used during this study in order to accommodate 32 striped bass (1.56 ± 0.07 g dry weight). The barriers segregated 4 PCB-exposed fish from 4 nonexposed fish in each tank. The tanks were equipped with air-driven filters containing 130 g activated carbon and a small piece of polyester fiber to trap waterborne particles.

The fish in the secondary uptake study received either two doses of ^{14}C-PCB in Gammarus followed by a third feeding of nonlabeled Gammarus (exposed), or three feedings of nonlabeled Gammarus (sham-exposed). The feeding interval was 48 hr. Gill, liver, gall bladder, alimentary tract spleen, heart, eyes, brain, head and remaining carcass were analyzed for ^{14}C-PCB. Feces were collected from both sides of the barrier tanks at 24-hr intervals; feces had become mixed, and were pooled for use in balancing ^{14}C-PCB masses. Holding water was taken for ^{14}C-PCB analysis 24, 72 and 120 hr after the start of the experiment.

Statistical analyses were performed as presented by Zar (1974). Percentage of administered dose retained by tissue compartments and whole fish were presented as the mean $\bar{x}$ plus or minus standard error ($s_{\bar{x}}$. Differences between group PCB levels at the different sampling times were tested by a one-way analysis of variance (ANOVA), with (P) $\leq$ 0.05. Where significant differences were found, a multiple range test

(Newman-Keuls) was performed to determine significant differences between all possible pairs of group means.

Ninety-five percent confidence intervals (C.I.) for the PCB absorption and elimination rate constants were determined from the slopes and standard errors of the least squares lines (Pizza and O'Connor, 1983).

Tissue compartment and whole fish PCB analysis was performed on sample replicates at each sampling time. To make the best use of the data, the least squares regressions used to estimate absorption and elimination rate-constants were performed with multiple values of Y for each X rather than by analysis of means. This permitted testing for linearity of regression. Analysis of covariance (ANCOVA) was performed to determine whether significant differences existed among the elimination rate constants.

RESULTS

The data presented here for the distribution of ^{14}C-PCB among various tissues and organs are expressed primarily as percent of the dose administered or percent of the total body burden. Mass balancing of administered dose was not attempted since some portion of the dose administered was removed from the aquaria by the carbon filters, or by removal of feces. The data for ^{14}C-PCB in feces and water (Table IV) show that the highest concentrations occurred 24 hr after feeding, a time which corresponds with clearance of the gut (Pizza, 1983). Feces produced after the period of gut clearance contained far lower concentrations of PCB (e.g., Table IV; single dose study, 48 to 96 hr; secondary uptake, 72 to 144 hr).

Table IV. Concentrations of ^{14}C-PCB in feces (as µg/g dry weight) and in holding water (as ng/ℓ) during the three striped bass studies. The listing of events of dosing with ^{14}C-PCB or sham dosing provides the experimental protocol. The data are given as the mean ± 1 standard error. Numbers of fish used for each determination are in parentheses.

	Single Dose			Multiple Dose			Secondary Uptake		
Time (hr)	Event	Feces (µg/g)	Water (ng/ℓ)	Event	Feces (µg/g)	Water (ng/ℓ)	Event	Feces (µg/g)	Water (ng/ℓ)
0	^{14}C-Dosing	-	-	^{14}C-Dosing	-	-	^{14}C-Dosing	-	-
6		-	0.35±0.07 (2)		-	-		-	-
24		4.83±1.52 (8)	0.55±0.09 (8)		4.01±0.62 (3)	0.53±0.14 (3)		2.18±0.44 (4)	0.46±0.07 (4)
48		0.66±0.24 (4)	-	^{14}C-Dosing	0.77±0.29 (3)	-	^{14}C-Dosing	0.08±0.03 (4)	-
72		0.36±0.20 (2)	0.23 (1)		5.76±1.41 (3)	0.55±0.15 (3)		1.73±0.16 (4)	1.00±0.14 (4)
96		0.15±0.04 (3)	0.18 (1)	^{14}C-Dosing	1.57±1.01 (3)	-	Sham Dosing*	0.32±0.21 (4)	-
120		-	-		3.86±0.39 (3)	0.26±0.09 (3)		0.14±0.01 (3)	0.19±0.05 (3)
144		-	-		0.51±0.20 (3)	-		0.16±0.03 (3)	0.13±0.09 (3)

* = ^{14}C-PCB free exposure; ( ) = n

Concentrations of ^{14}C-PCB in the holding water were correlated with PCB concentrations in feces in the single dose and multiple dose studies ($y = -1.21 + 11.40\ x$; $r^2 = 0.74$). This suggests that water column concentrations of PCB were due in great part to dissolution of PCB from feces into the water. Regression analysis was not performed on the data from secondary uptake studies, since feces from exposed and unexposed fish became mixed.

The single dose study provides a record of distribution of PCB among tissues during both the assimilation phase and the elimination phase. Except for the special case of the alimentary tract, the greatest portion of the administered dose occurred in each tissue between 24 and 48 hr after feeding (Table V). Except for the gill, PCB content in each tissue decreased from the maximum until the experiment was terminated at 120 hr. Approximately 47% of the administered dose remained after 120 hr (Table V).

Transport of PCB to tissue compartments was rapid; after only 6 hrs, about 15% of the administered dose had been distributed among the gill, liver, head and body musculature (carcass) (Table V); 5-7% of the dose had been lost, presumably due to excretion across the gill surface, since the holding water at 6 hr was already carrying a measurable quantity (0.35 ng/L) of ^{14}C-PCB.

PCB elimination rates for body compartments were determined from data gathered after the maximum absorption from the PCB source (after 24 hr). The tabulated values for each compartment were log-transformed for least squares linear regression analysis. The slopes of the regression lines were used to determine the elimination rate-constant (k_e) in hr^{-1}

Table V. Distribution of ^{14}C-PCB among tissue and organ compartments after administration of a single dose of 0.5 µg ^{14}C-PCB at time 0. All data are presented as a percentage ($\bar{x} \pm s_{\bar{x}}$) of the dose administered.

Time from Administration (hr)	Gill	Liver + Gallbladder	Alimentary Tract	Head	Remaining Carcass	Whole Fish	n
0	-	-	100.00±1.89	-	-	100.00±1.89	10
6	1.00±0.18	2.06±0.45	76.54±2.11	4.41±0.04	9.21±0.77	93.21±2.37	10
12	1.39±0.22	3.68±0.78	44.70±5.49	10.89±1.60	23.11±3.07	83.73±2.29	10
24	4.65±0.86	4.01±0.59	7.03±0.39	-	-	87.33±1.27	3
48	1.96±0.43	4.53±0.44	4.08±0.26	22.10±1.86	44.99±4.27	77.65±6.38	5
72	1.49±0.15	3.70±0.18	3.88±0.48	21.73±2.16	36.04±3.07	66.85±5.45	5
96	2.14±0.19	2.68±0.19	3.54±0.20	19.72±1.06	32.44±1.72	60.51±2.35	10
120	1.44±0.14	2.89±0.34	2.87±0.31	15.63±2.09	24.17±1.81	46.99±4.29	5

from the equation $\log x = \log x_o - k_e t/2.3$; half-lives of the compound in the different compartments were determined from the rate-constant as $(-\ln 0.5/k_e)$ (Goldstein et al., 1974; see also Pizza and O'Connor, 1983).

The PCB elimination data showed linearity of regression for every compartment tested. The slopes of the line were significantly different from zero for all compartments except the gill. The regression lines are presented in Fig. 6. PCB levels in gill fluctuated throughout the experiment; the slope of the regression line was not significantly different from zero.

The liver/gall bladder compartment showed a steady increase from 2.01% to 4.5% of the administered dose by 48 hr. From this point, the quantity declined to 2.9% at 120 hr. The mean PCB burdens carried by gall bladder relative to the total for this compartment (liver and gall bladder) were 8.9 ± 1.5, 11.1 ± 2.4, and 22.3 ± 3.1% at 6, 12, and 96 hr, respectively. The relative PCB burden in the gall bladder at 96 hr was significantly greater than at both 6 and 12 hr, showing PCB movement from liver to gall bladder sometime after 12 hr.

Liver and gall bladder, when grouped as a single compartment (Table V), showed PCB elimination with $k_e = 0.0076\ hr^{-1}$; the 95% CI was from 0.0059 to 0.0093 hr^{-1}. The calculated half-life for PCB in the liver/gall bladder compartment was 91.2 hr.

The quantity of PCB in the alimentary tract showed a rapid reduction. The mean level showed a decline at every sampling time with 2.87 ± 0.31% of the initial PCB dose remaining at 120 hr. The first 30 hr has

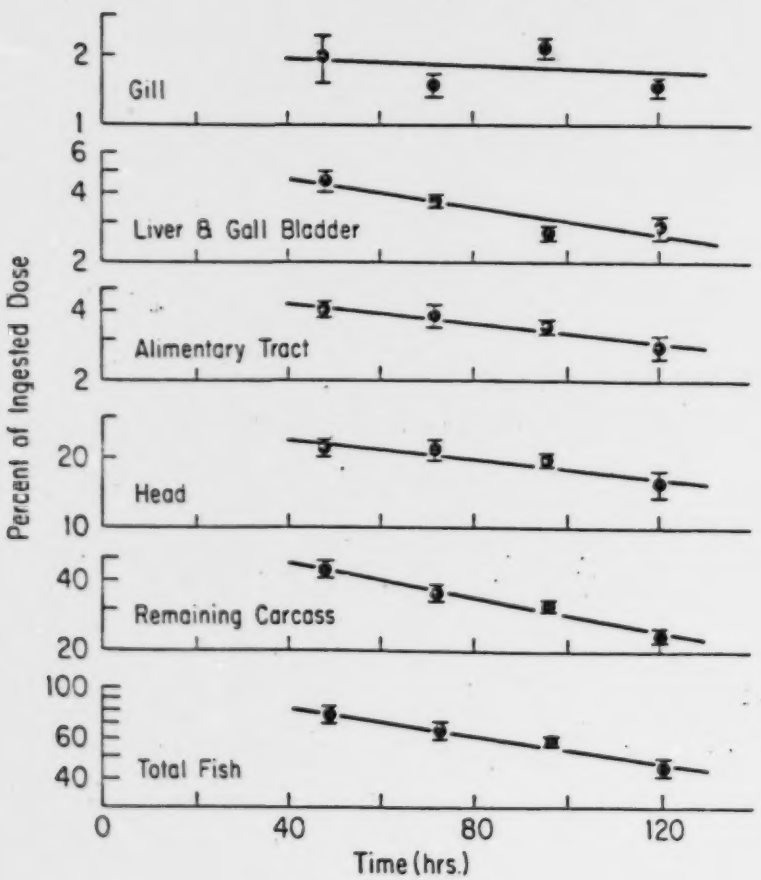

Gill
Liver & Gall Bladder
Alimentary Tract
Head
Remaining Carcass
Total Fish
Percent of Ingested Dose
Time (hrs.)
0
40
80
120

Figure 6.

been defined as the source absorption phase; the uptake rate was discussed in Pizza and O'Connor (1983).

The head (minus gill) was treated as a compartment separate from the remaining body. PCB elimination from this site occurred with a 95% C.I. for the rate constant equal to 0.0032 to 0.0062 hr^{-1} and corresponding half-life of 147.7 hr. PCB levels in brain were determined separately at 6, 12, and 96 hr. The mean levels in brain relative to the total burden in head (minus gill) were 12.5 ± 1.2, 11.7 ± 1.4, and 13.4 ± 1.6%, respectively. These levels were not significantly different, indicating that PCB elimination from the whole head is representative of brain.

The PCB elimination rate was determined for the remainder of the fish which will be referred to as carcass (whole fish minus gill, liver, gallbladder, alimentary tract, and head), pooled as a single compartment. The regression line yielded a 95% C.I. for the k_e equal to 0.0066 to 0.0092 hr^{-1}, corresponding to a half-life of 87.8 hr.

The PCB elimination rate constant for the whole body, calculated from the time of oral administration to 120 hr, was 0.0054 hr^{-1} (95% CI = 0.0046 to 0.0062 hr^{-1}). When the regression was performed only for data after maximum absorption, the 95% C.I. for the whole body k_e was 0.0053 to 0.0077 hr^{-1}. These two intervals were not different. Analysis of covariance for the regression lines of all compartments tested (other than gill) showed that the elimination rate constants of the different tissue compartments of this study were not significantly different.

During the multiple-dose study, the distribution of PCB in striped

bass was determined for one, two, and three ^{14}C-PCB doses. Each dosage was 387 ± 13 ng and they were given at 48 hr intervals. The results of this study are presented as percentage of the retained ^{14}C-PCB, dry weight tissue concentration (μg PCB/g), and as percentage of the cumulative dose found in tissues and the remaining body (Table VI). During the multiple dose study, spleen and heart were separated from the carcass compartment and analyzed separately for PCB.

Forty-eight hours after the first dose was administered, the majority of the PCB retained was found in head (28.5%) and carcass (57.1%). Gill, liver/gall bladder, alimentary tract, and spleen/heart each carried 6% or less of the body burden. Comparing the percentage of cumulative dose in this experiment to the results of the single dose study (Table V), the reader can see that the data are essentially the same. This verifies tissue distribution data for the single-dose study and provides a replication of the experimental procedure.

The PCB tissue distribution pattern did not change during the two subsequent intervals (Table VI). Among the three levels of exposure (1, 2 and 3 doses), the percentage of the whole-body ^{14}C-PCB burden retained by each compartment did not differ significantly.

Tissue burdens presented as a percentage of the cumulative dose show a decrease in mean level with increased dosing for all compartments analyzed, including whole fish. This decrease is the result of elimination of administered PCB during the interval.

The PCB concentration data (Table VI) show a stepwise increase for ^{14}C-PCB in the tissues for each of the successive doses. For example,

Table VI. Distribution of ^{14}C-PCB among tissue and organ compartments measured 48 hr after administration of 1, 2, or 3 doses of PCB. Each dose was ∿ 387 ng ^{14}C-PCB. All the data are presented as $\bar{x} \pm s_{\bar{x}}$.

Doses Given		Gill	Liver + Gallbladder	Alimentary Tract	Spleen + Heart	Head	Carcass	Epaxial Muscle	Whole Fish
	Percent of retained burden	2.47 (±0.38)	5.94 (±0.66)	5.35 (±0.41)	0.57 (±0.08	28.54 (±1.00)	57.14 (±1.38)	-	100
One (n=5)	µg PCB/g (dry)	0.33 (±0.06)	1.51 (±0.17)	0.54 (±0.06)	0.34 (±0.06)	0.41 (±0.04)	0.32 (±0.03)	0.26 (±0.04)	0.37 (±0.04)
	Percent of cumulative dose	1.92 (±0.42)	4.45 (±0.43)	4.00 (±0.25)	0.42 (±0.04)	21.70 (±1.82)	46.14 (±5.40)	-	76.24 (±6.26)
	Percent of retained burden	2.44 (±0.18)	6.12 (±0.88)	5.64 (±0.15)	0.58 (±0.11)	30.11 (±1.12)	55.11 (±1.90)	- -	100
Two (n=3)	µg PCB/g (dry)	0.53 (±0.10)	2.98 (±0.23)	1.10 (±0.11)	0.95 (±0.13)	0.69 (±0.15)	0.54 (±0.09)	0.58 (±0.08)	0.63 (±0.11)
	Percent of cumu- dose	1.61 (±0.26)	3.89 (±0.33)	3.66 (±0.34)	0.36 (±0.04)	19.48 (±1.40)	36.21 (±4.92)	- -	65.23 (±6.90)
	Percent of retained burden	2.10 (±0.22)	6.15 (±0.34)	6.48 (±1.11)	0.56 (±0.04)	27.61 (±0.41)	57.09 (±1.82)	- -	100
Three (n=5)	µg PCB/g (dry)	0.74 (±0.07)	4.47 (±0.58)	1.73 (±0.16)	0.79 (±0.04)	1.01 (±0.08)	0.87 (±0.07)	0.85 (±0.07)	0.98 (±0.08)
	Percent of cumulative dose	1.25 (±0.18)	3.63 (±0.31)	3.83 (±0.71)	0.34 (±0.04)	16.25 (±0.82)	33.60 (±2.08)	- -	58.91 (±3.28)

levels of 0.37, 0.63 and 0.98 µg PCB/g were measured in whole fish for the first, second, and third feedings, respectively. The liver/gall bladder compartment had the highest PCB concentration of all compartments for all three exposures (1.51 ± 0.17, 2.98 ± 0.23, and 4.47 ± 0.58 µg/g, respectively). For each level of exposure (1, 2, or 3 doses), the PCBs in the liver/gall bladder compartment were significantly greater than in the other tissues analyzed in both concentration and rate of increase with dose. There were no significant differences among the other compartments. The concentration data show the overall increase in mean levels with increased dosing.

The secondary uptake data (Table VII) showed that the amount of ^{14}C-PCB in the fish's body due to accumulation of material directly from water was 2-3% of the total burden retained. The proportion of the secondary body burden was also calculated for each compartment (Table VII). With the exception of the alimentary tract data, the percent body burden in each compartment agreed quite closely with the burdens due to dietary uptake alone. The apparently anomalous data for the alimentary tract are probably due to the fish in the control chambers ingesting PCB-contaminated feces.

DISCUSSION

These studies of the fate and distribution of PCBs among tissue compartments in striped bass show evidence of rapid and dynamic movement from the site of absorption to the tissue. Further, it can be seen that there exist tissue-specific differences in PCB concentration, and that the major route for PCB elimination is via the hepatic pathway.

Table VII. Results of the secondary uptake studies in which the non-dosed fish were exposed only to ^{14}C-PCB in water after elimination from fish receiving two doses of ^{14}C-PCB in food. The data are presented as the mean of percent of total dose retained, ng of ^{14}C-PCB retained, percent of retained body burden in tissue, and the percent tissue burden in non-dosed fish compared to dosed fish.

	% total dose (980 ng) in tissue		^{14}C-PCB mass (ng) in tissue		% retained dose in tissue		% ng ^{14}C-PCB retained Non-dosed/ Dosed
	Dosed	Non-dosed	Dosed	Non-dosed	Dosed	Non-dosed	
Gill	4.32	0.06	42.34	0.59	5.84	4.00	1.40
Liver & gall bladder	3.33	0.07	32.63	0.69	4.50	4.66	2.12
Alimentary tract	6.86	0.14	67.23	1.37	9.28	9.26	2.04
Spleen & heart	0.30	n.d.	2.94	n.d.	0.41	-	-
Head	15.42	0.29	151.12	2.84	20.86	19.19	1.88
Carcass	43.69	0.95	428.16	9.31	59.10	62.90	2.17
Whole fish	73.92	1.51	724.42	14.80	-	-	2.04

The combined results of the single dose and multiple dose study demonstrate that the onset of PCB assimilation occurs within six hours of feeding, and that distribution of the first and subsequent doses among tissue compartments in the striped bass is uniform. In a physiological sense, therefore, the fate of PCBs ingested by fishes is similar to that found for drugs in general (Goldstein et al., 1974) and for PCBs in particular (Morales et al., 1979) among mammals.

The consistency with which the administered dose of PCB was distributed among the tissue compartments in striped bass also imposes a gradual increase in body burden with exposure. Such appears to be the case whether fishes are exposed to PCB in the diet (as in this study) or in the water (Branson et al., 1975; Mayer et al., 1977; Califano et al., 1982). While the tissue distribution of PCBs is apparently not affected at low level exposures, it remains to be determined whether the same patterns hold true for high-level exposures in heavily contaminated environments. It is possible that compartments may become overloaded with high level exposure, and that tissue distributions would be altered, thus leading to adverse physiological effects. Such data are not available in the literature.

We speculate, however, that for PCB burdens which occur among fishes in the heavily contaminated Hudson River and estuary, tissue distribution of PCB remains similar over concentrations of more than two orders of magnitude. Available data on three species (Morone saxatilis, M. americana and Trinectes maculatus) suggest that the proportions of PCB in muscle, liver and other tissues are similar whether body burdens in nature are 0.5 µg/g (dry weight) or 40 µg/g (dry weight). That over-

loading of tissue compartments and toxic effects apparently does not occur for PCBs in fishes is supported by the current absence of data to support a relationship between body burdens and physiological effect in natural populations of estuarine and marine fishes.

Except for the liver/gall bladder compartment, PCB distribution to striped bass tissue was proportional to the mass of tissue. Califano (1981) suggested that this was related to the volume and rate of arterial blood supply to each tissue compartment. For dietary exposures, the liver may be expected to accumulate increased levels of PCB since the hepatic portal circulation may carry a large portion of the assimilated dietary burden directly to the liver. In the current study the liver/gall bladder compartment contained PCBs at levels about four times higher than in all other body compartments. The same relationship is generally found in environmental samples of marine fishes (Boehm and Hirtzer, 1982; MacLeod et al., 1981) and in laboratory studies of direct water uptake (Califano, 1981; Pizza, unpublished data). Since direct water uptake of PCB results in the same magnitude of PCB accumulation in the liver as dietary exposure (Califano, 1981), hepatic portal circulation cannot be the full explanation for increased liver burdens. Rather, the affinity of PCB for liver tissue must be associated either with the role of liver in biotransformation of nonpolar compounds, or with the high lipid content of liver tissue in fishes.

The rate-constants for PCB elimination from the various tissues measured were, except for the gill, quite similar. This demonstrates that partitioning to lipid in tissues was probably not occurring in the experiments conducted here. It also demonstrates that the affinity of

liver for PCB is due more to metabolic function than partitioning to a chemical or structural entity unique to liver tissue. Indeed, since the elimination rate constants for all tissues except the gill were similar, and since liver PCB concentrations were greater than other tissues by a factor of about 4, then it follows that PCB turnover rate in the liver is about four times greater than in other striped bass tissues.

Whether these facts can be used to estimate the mass of PCB eliminated from the fish via the hepatic pathway remains unclear. In studies of PCB elimination by mammals and birds, investigators have documented the presence of PCBs or PCB metabolites in urine and fecal material (Hutzinger et al., 1972; Morales et al., 1979). In a study by Morales et al. (1979), the PCB was given either by intraperitoneal injection or in drinking water. In both cases the PCB was present in feces, clearly demonstrating the hepatic-bile pathway for PCB elimination. Melancon and Lech (1976) found PCB in the bile of fishes fed PCB-contaminated food. In the present study, PCB in the bile increased two-fold by 96 hr after feeding. Presumably, PCB in bile will be excreted to the intestine for eventual elimination with feces.

It has not been demonstrated, however, to what extent PCBs in bile may be reabsorbed in the gut of fishes. Borgstrom (1974) suggests that lipids, triglycerides and hydrocarbons present in the diet of fishes require emulsification by bile prior to absorption across the intestinal surface. Bile-associated PCBs, therefore, may be partially reabsorbed in the intestine. Certainly some portion is excreted in feces, as shown by the PCB concentrations in fecal matter observed in our studies, but the matter of the magnitude of the hepatic pathway in PCB elimination in

fishes certainly deserves further study.

The question of PCB elimination across the gill has been demonstrated by other workers (Califano, 1981; Guiney et al., 1977). The results of this study show that there was essentially no loss of PCB (as percent dose) from the striped bass gill tissue over the 120 hr elimination study. As with the other tissue compartments, the concentration of PCB in the gill increased when three successive doses of PCB were given. It is possible that throughout all the experiments the striped bass gill tissue remained in dynamic equilibrium with the water in the aquaria. Due to continuing PCB input from other tissues, combined with gradual loss to the water across the gill, we may have been unable to detect changes in either PCB concentration or proportion of administered dose in the gill.

The results for young-of-year striped bass indicate that for extremely low level PCB exposure (< 1.0 µg), the whole-body distribution of PCB is not affected by an increase in burden. Since the lipid content varies from compartment to compartment in fishes (Lieb et al., 1974; Gruger et al., 1975), the possibility exists that the lipid-mediated distribution of organochlorine contaminants may be secondary to an initial low level situation where PCB partitioning is determined by a tissue constituent which is more uniformly distributed than lipid. Indeed, lipid content is unlikely to be the only determinant of distribution since discrepancies exist between lipid and PCB content of certain tissues (Peterson and Guiney, 1979). Movement of PCB to lipid possibly occurs only after the primary site is saturated. This is completely conjectural, and studies on the effects of body burden and lipid

content are needed before distribution can be more fully understood.

Past studies have determined the elimination rates of different organs/tissues in an attempt to discern which were representative of whole body (Guiney et al., 1977; Guiney and Peterson, 1980). Since it is technically unfeasible to quantitatively analyze whole fish of more than several grams dry weight, a tissue having a rate-constant corresponding to whole-body elimination would be of great value when working with adult fish.

It is a general finding that PCB is absorbed and eliminated in some proportion to the lipid content of the tissue (Hamelink et al., 1971; Lieb et al., 1974). Guiney et al. (1977) found that for all body compartments studied, only bile and blood were not representative of the whole-body PCB elimination rate.

The results of the current work show that for the tissues analyzed, all except gill gave a valid estimate of the whole-body elimination rate-constant. Some of the tissue, however, would not be recommended as the focus of future work. Obviously, gill PCB accumulation is too dependent on the concentration of the holding water. Other compartments, namely gall bladder and brain, are sufficiently difficult to remove from the body without rupture and/or cross-contamination during dissection that they should not be the sample of choice.

The majority of research of PCB accumulation in fish suggests that if determination of the whole-body elimination rate is the ultimate goal (disregarding body distribution), this should be accomplished with uniform subsamples of whole body.

VII. ECOKINETIC MODEL FOR PCB ACCUMULATION IN FISHES

INTRODUCTION

Contaminant loads to the marine ecosystem adjacent to New York and New Jersey have been well documented (Mueller et al., 1982). Among the more important are the polychlorinated biphenyls (PCBs), due primarily to their abundance in the Hudson-Raritan system (Bopp et al., 1981; O'Connor et al., 1982), their toxicity (National Academy of Sciences, 1979), and their potential to cause chronic effects in animal and human populations (Kuratsune, 1976; Mehrle et al., 1982).

Most of the PCB contamination in the New York Bight derives from ocean dumping of sewage sludge and waste dredged material (Table VIII). When relative PCB contribution from direct discharges is considered and integrated according to typical flow patterns in the Bight, expected water concentrations should be greatest in the vicinity of the N.Y. Bight ocean disposal sites. Actual data from a variety of studies shows this to be the case. We have calculated that the elevated PCB levels near the New York Bight dumpsites derive in roughly equal portions from dredged material and sewage sludge. The increased PCB levels in the water column increase the potential for PCB uptake in all trophic levels of the Bight ecosystem (Wyman and O'Connors, 1981; Califano et al., 1982; Brown et al., 1982).

The majority of PCB placed in the Bight system with dredged

Table VIII. Estimated PCB inputs to the N.Y. Bight Apex, in Kg/year.

Source	Max.	% Total	Min.	% Total
Atmospheric[a]	490	7	34	1
Municipal Wastewater[b]	42	0.6	42	1
Dredged Material[c]	3500	51	1800	61
Sewage Sludge[d]	1300	19	750	26
Hudson Plume[e] (part.)	1037	15	62	2
Hudson Plume (dissolved)	480	7	240	8
Totals	6849	996	2928	99

[a] Assumes 1.14 m/year precipitation at 15 (min) and 215 (max) ng/ℓ PCB.

[b] 99.1 MGD direct wastewater flow; all secondary at 0.3 µg/ℓ PCB.

[c] From Bopp et al. (1981; min) and O'Connor et al. (1982; max).

[d] Based upon estimates from West and Hatcher (1980), Bopp et al. (1981) and O'Connor et al. (1982).

[e] Plume flow assumed to be 6.6×10^{10} ℓ/day, carrying 3 (min) and 50 (max) mg/ℓ solids at 0.86 µg/ℓ PCBs for particulate load, and 10 ng/ℓ (min) and 20 ng/ℓ (max) for dissolved load. (See Mueller et al., 1982).

materials remains associated with deposited particulates. Coring studies at the dredged material dumpsite in the New York Bight (NYUMC, 1982) show that PCB levels vary with depth of core. Ditoro et al. (in press) have shown that PCB mobilization from deposited sediments is slow; vertical migration is estimated to be on the order of millimeters per year. Thus, the contribution of dredged material to PCB levels in the New York Bight water column is associated primarily with losses which occur during the dumping process. Tavoloro (1982) has estimated a dry mass loss of ~ 4% during dumping. Overall PCB losses during the dumping process may be on the order of 15%.

With good reason, it has been concluded that activities which contribute to PCB levels in N.Y. Bight fishes should be minimized. In an attempt to determine how dredged material dumping affects PCB body burdens in fishes, several investigations have evaluated the rates and routes of PCB transport in marine ecosystems (O'Brien and Gere, 1979; Rubinstein et al., 1983. Once transport mechanisms are understood, predictive models may be formulated regarding the extent to which PCBs in sediment may cause increased body burdens. If unacceptable PCB burdens in marine organisms (e.g., 1 μg/g, 5 μg/g) can be related to mud dumping and the process of accumulation is well understood, managers and regulators can take steps to reduce or eliminate the problem.

Since the early 1970's it has been thought that fishes accumulated PCB directly from water (Hamelink et al., 1971; Neely et al., 1974). Experimentally derived bioconcentration factors (BCF) predictive of "steady state" burdens in fishes have been published widely (Table IX); uptake mechanisms based upon octanol-water partition coefficients and lipid solubility of PCB have been proposed (i.e., the equilibrium partition theory; Neely et al., 1974; Branson et al., 1975; Mackay, 1982). The same mechanisms have been proposed to explain PCB accumulation in zooplankton (Clayton et al., 1977; Pavlou and Dexter, 1979).

Several factors mitigate against the equilibrium partition theory as the full explanation for PCB burdens in marine fishes. First, the concept was developed using pure, dissolved compounds in relatively particle-free water. Under natural circumstances sea water contains many particles to which PCBs are likely to sorb (Hiraizumi et al., 1979; Nau-Ritter et al., 1982). For striped bass (Morone saxatilis), Califano et al. (1982) showed that the presence of particles in bioassay water decreased the quantity of PCB available for uptake, and that body burden was directly related to "available" PCB rather than total PCB.

Second, published BCF data generally derive from experiments of long duration, $\geq$ 5 days. Under such circumstances, organisms must be fed during the test, and the proportion of the PCB

Table IX. Bioconcentration of various Aroclors in fish.

Organism	Commercial Aroclor Mixture	Exposure Concentration ($\mu g\ \ell^{-1}$)	Exposure Time (days)	BCF[a]	Reference
Channel catfish (*Ictalurus punctatus*)	1248	5.8	77	5.6×10^4	Mayer et al., 1977
	1254	2.4	77	6.1×10^4	"
Bluegill sunfish (*Lepomis macrochirus*)	1248	2-10	chronic	2.6 to 7.1 $\times 10^4$	Stallings and Mayer, 1972
	1254	2-10	chronic		
Brook trout (fry) (*Salvelinus fontinalis*)	1254	6.2	118	4.6×10^4	Mauck et al., 1978
Spot (*Leiostomus xanthurus*)	1254	1	56	3.7×10^4	Hansen et al., 1971
Pinfish (*Lagodon rhomboides*)	1016	1	56	1.7×10^4	Hansen et al., 1974
Rainbow trout (*Salmo gairdneri*)	2,2',4,4'-tetrachloro-biphenyl	1.6 and 9.0	5	2.9×10^4	Branson et al., 1975
Fathead minnow (*Pimephales promelas*)	1248	3.0	250	1.2×10^5	DeFoe et al., 1978
	1260	2.1	250	2.7×10^5	"

[a] Bioconcentration factor determined from the concentration in the fish divided by the concentration in the exposure water.

accumulated with contaminated food was not accounted for in these designs. Many studies have shown that food material, both living and dead, accumulates PCB rapidly (Wyman and O'Connors, 1981; Peters and O'Connor, 1982), providing a secondary route of PCB uptake in the test chamber. Peters and O'Connor (1982), for example, showed that the common striped bass food organisms Gammarus and Neomysis accumulated up to 2 µg/g PCB from water in less than 10 hours exposure to a concentration of 1 µg/L. Inanimate food may accumulate PCB just as rapidly (Wyman and O'Connors, 1981). Thus, BCF values calculated from long-term exposures include a dietary uptake component not accounted for in application of the data to equilibrium partitioning theory.

Third, and perhaps most importantly, equilibrium partition calculations for PCB bioconcentration generally yield estimates which are low, relative to field observations (Table X). Given the potential importance of PCB as a toxicant in natural systems, we feel it is unwise to rely heavily upon such "order-of-magnitude" estimates. Given the knowledge that all parts of the marine food web contain PCB (MacLeod et al., 1981; O'Connor et al., 1982), and that cross-gut assimilation of PCB in fishes approaches 90% (Pizza and O'Connor, 1983), we stress that PCB in fishes derive in some significant part from the food. Recently published models for contaminant transport in fishes and plankton demonstrate that dietary sources may be the prime determinant of

Table X. Calculation of expected PCB body burdens in fishes from equilibrium partitioning based upon New York Bight data.

	Min	Max
Water column PCB concentration (ng/ℓ)[a]	10	40
Particulate/dissolved ratio[b]	0.67	0.67
Dissolved (available) PCB (ng/ℓ)	6.7	27
Bioconcentration factor[c]	$1x10^4$	$1x10^4$
Expected concentration (µg/g fish)[d]	0.07	0.27
Observed concentrations (µg/g)[e]		
Striped bass	0.6-3.8	
Winter flounder	0.1	
Atlantic mackerel	0.5-0.7	
Bluefish	0.7-3.6	
American eel	0.5-0.8	
Tautog	0.6	

a Concentrations derived from Lee (1977), Lee and James (1978), IEC (1979), Pequegnat et al. (1980) and MacLeod et al. (1981).

b Various authors suggest particulate/dissolved PCB ratios ranging from zero to about 1. Based upon suggested values from Brown et al. (1982), Nau-Ritter (1980) and Pavlou and Dexter (1979) we arrived at a value wherein two-thirds of the total water column PCB may be in the dissolved state.

c Based upon chronic bioassay data wherein values range from $1.7x10^4$ to $6.1x10^4$ (Table 2) for various species. Assuming some fraction to be associated with feeding, we suggest $1x10^4$ to be a reasonable and conservative BCF approximation (see text).

d Calculated as (g/g PCB in water) x BCF, wherein the water value equals ng ($g \times 10^{-9}$) ÷ 1030, the weight of one liter of sea water at 30 parts per thousand salinity.

e Data from O'Connor et al., 1982; N.Y. State DEC, 1981; 1982; N.J. Dept. of Environmental Protection, 1982.

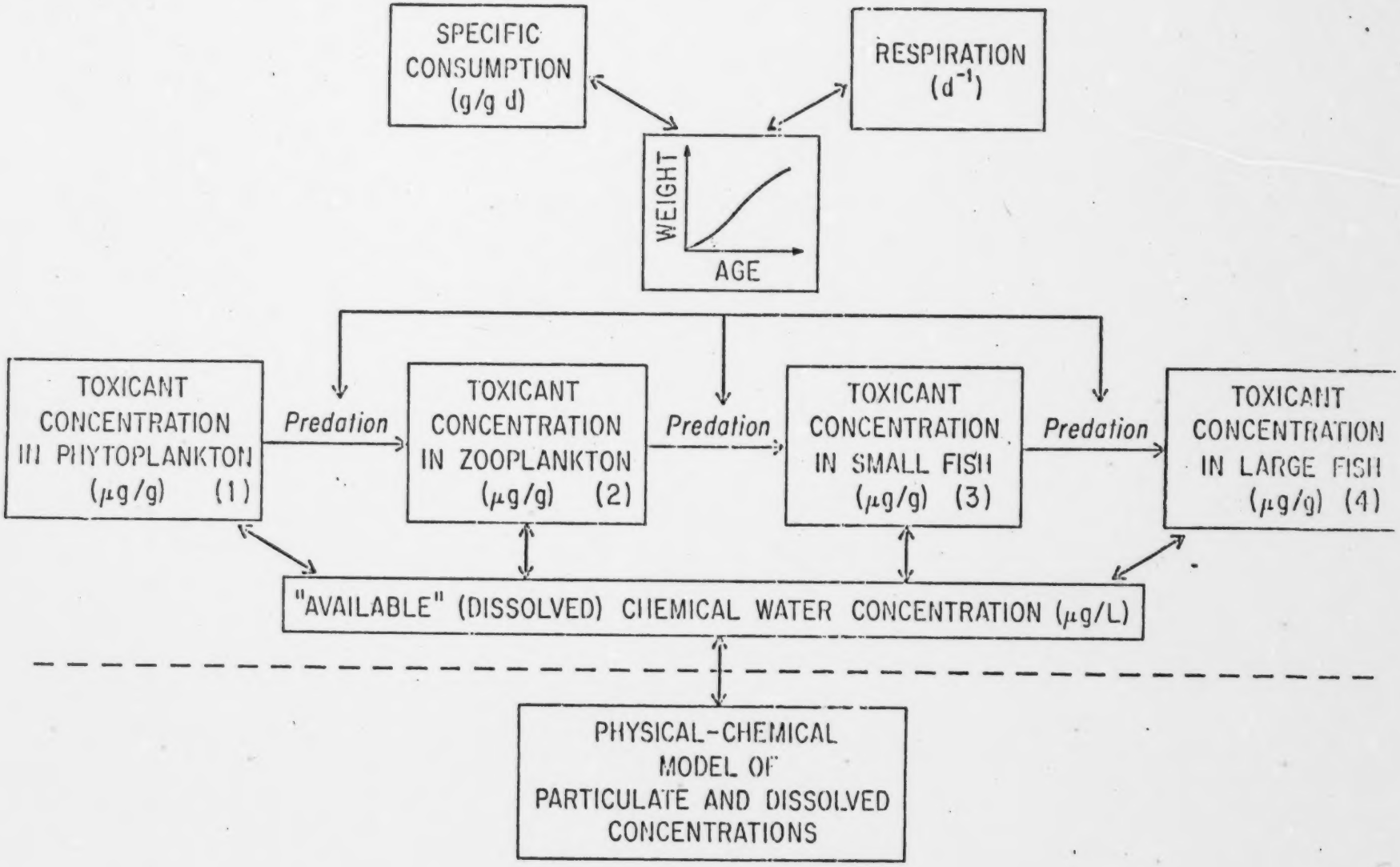
PCB body burdens (Thomann, 1981; Brown et al., 1982) (Fig. 1).

Studies of PCB uptake from diet have been carried out in our laboratory for a variety of fishes and crustaceans (Califano et al., 1980, 1982; Califano, 1981; Pinkney, 1982; Pizza, 1983; Pizza and O'Connor, 1983). The remainder of this paper provides the results of our food chain transport studies of PCB in marine fishes, principally striped bass. The concepts presented here are based upon pharmacokinetic relationships (Goldstein et al., 1974). Pharmacokinetics have been applied to questions of pesticide uptake in fishes by Krzeminski et al. (1977) and similar principles underlie the recent work of Thomann (1981) in his efforts to model PCB and cadmium accumulation in fishes. We propose here a seasonally variable, pharmacokinetic model to predict dietary accumulation of PCB in fishes.

METHODS

The techniques used in these studies were developed by Califano et al. (1982) and by Pizza (1983). Complete details of the pharmacokinetic approach to PCB studies are available in Pizza (1983; see also Pizza and O'Connor, 1983). To summarize, an evaluation of the assimilation and excretion of a compound required empirical data as to the rate of assimilation from the absorption site (k_a), and the rate of loss of the compound (k_e) from some physiological pool. The pool we used was the entire

Fig. 7. Schematic of ecological and physiological factors influencing PCB accumulation in fishes. Note the separation of food and water sources, and the proposed impact of physiological factors (feeding, respiration, metabolism) on food chain transport. Adapted from Thomann, 1981.


SPECIFIC CONSUMPTION (g/g d)
RESPIRATION (d^{-1})
WEIGHT
AGE
TOXICANT CONCENTRATION IN PHYTOPLANKTON (μg/g) (1)
Predation
TOXICANT CONCENTRATION IN ZOOPLANKTON (μg/g) (2)
Predation
TOXICANT CONCENTRATION IN SMALL FISH (μg/g) (3)
Predation
TOXICANT CONCENTRATION IN LARGE FISH (μg/g) (4)
"AVAILABLE" (DISSOLVED) CHEMICAL WATER CONCENTRATION (μg/L)
PHYSICAL-CHEMICAL MODEL OF PARTICULATE AND DISSOLVED CONCENTRATIONS

body mass of the subject organism. Pizza and O'Connor (1983) demonstrated that most body compartments have k_a and k_e values similar to the "whole body". The mathematics are straightforward, and based upon the exponential expression for a decay curve:

$$M = M_o e^{-k_a t} \text{ (for absorption)}, \qquad (1)$$

where M_o is the quantity of PCB placed at the absorption site, M is the quantity remaining at time = t, and k_a is the absorption rate constant.

Elimination rate constants (k_e) are derived from the same expression with different notation:

$$X = X_o e^{-k_e t}, \qquad (2)$$

where X_o is the whole body PCB level at time zero, X is the whole body PCB burden at time t, and k_e is the elimination rate constant.

Values for PCB assimilation were determined by force-feeding striped bass known quantities of ^{14}C-labeled PCB (Aroclor 1254) in natural food, and sampling at fixed intervals to determine: 1) the quantity of PCB in the gut; 2) the quantities of PCB in the whole body; and 3) the quantities of PCB in fecal material. Sampling of fishes continued for a period of 120 hours. Tissues were analyzed

for total ^{14}C-PCB by liquid scintillation counting (Pizza, 1983).

Experiments were conducted to determine k_a and k_e from single feeding and multiple feedings. Manipulation of the empirical data conformed to treatments suggested by Goldstein et al. (1974). The equations are documented in Table XI.

RESULTS

When striped bass were given single doses of PCB, there occurred initial and rapid elimination from the alimentary tract followed by a phase of less rapid elimination (Fig. 8A). Whole body elimination remained monophasic (Fig. 8B). Note that, after 48 hr, when alimentary tract burdens were < 10% of the dose, the whole body burden was high, reflecting nearly complete assimilation of PCB from the natural food matrix. For a single PCB dose, the body burden will follow a time course reflective of the ratio k_e/k_a, as shown in Fig. 9.

Fishes in contaminated environments, however, do not accumulate PCB as single, isolated dietary doses. Rather, they contain PCBs derived from water uptake, and they receive multiple, sequential doses of PCB in food. Our multiple dose study showed the gradual approach to "plateau" PCB levels (Fig. 10) expected for young striped bass exposed to sequential doses of PCB in food, given at 48 hr intervals. The interval was chosen in order to

Table XI. Pharmacokinetic expressions applied to PCB dietary uptake studies.

A. Absorption rate (k_a)	$M = M_o e^{-k_a t}$
B. Elimination rate (k_e)	$X = X_o e^{-k_e t}$
C. Absorption half-time $t_{\frac{1}{2}}$	$t_{\frac{1}{2}} = -\frac{-\ln 0.5}{k_a}$
D. Fractional absorption-single dose	$X/M_o = [e^{-k_e t/(k_e/k_a)} - e^{-k_e t}]/[(k_e/k_a)-1]$
E. Time to maximum absorption-single dose ($k_a \neq k_e$)	$t_{max} = [2.3 \log(k_a/k_e)]/(k_a-k_e)$
F. Maximum fraction absorbed-single dose	$X_{max}/M_o = (k_a/k_e)^{k_a/(k_e-k_a)}$
G. Body burden at end of dosing interval - multiple doses	$X = X_o e^{-k_e t^*} + X_o (e^{-k_e t^*})^2 + \ldots + X_o (e^{-k_e t^*})^n$
H. Body burden after dosing-multiple doses	$X_n = X_o (1-e^{-k_e t^* n})/(1-e^{-k_e t^*})$
I. Plateau (steady state burden-multiple dose	$X_\infty = X_o/1-e^{-k_e t^*}$
J. Fraction of plateau at "n" doses	$f = 1-e^{-k_e t^* n}$

Fig. 8 Experimental determination of PCB in striped bass after force-feeding. A. Loss of PCB from the alimentary tract showing a two-phase elimination. Phase 1 describes assimilation into the whole body. Phase 2 describes elimination in parallel with other tissues. B. Whole-body elimination of PCB. Note the similarity of slope with Phase 2 of alimentary tract elimination.

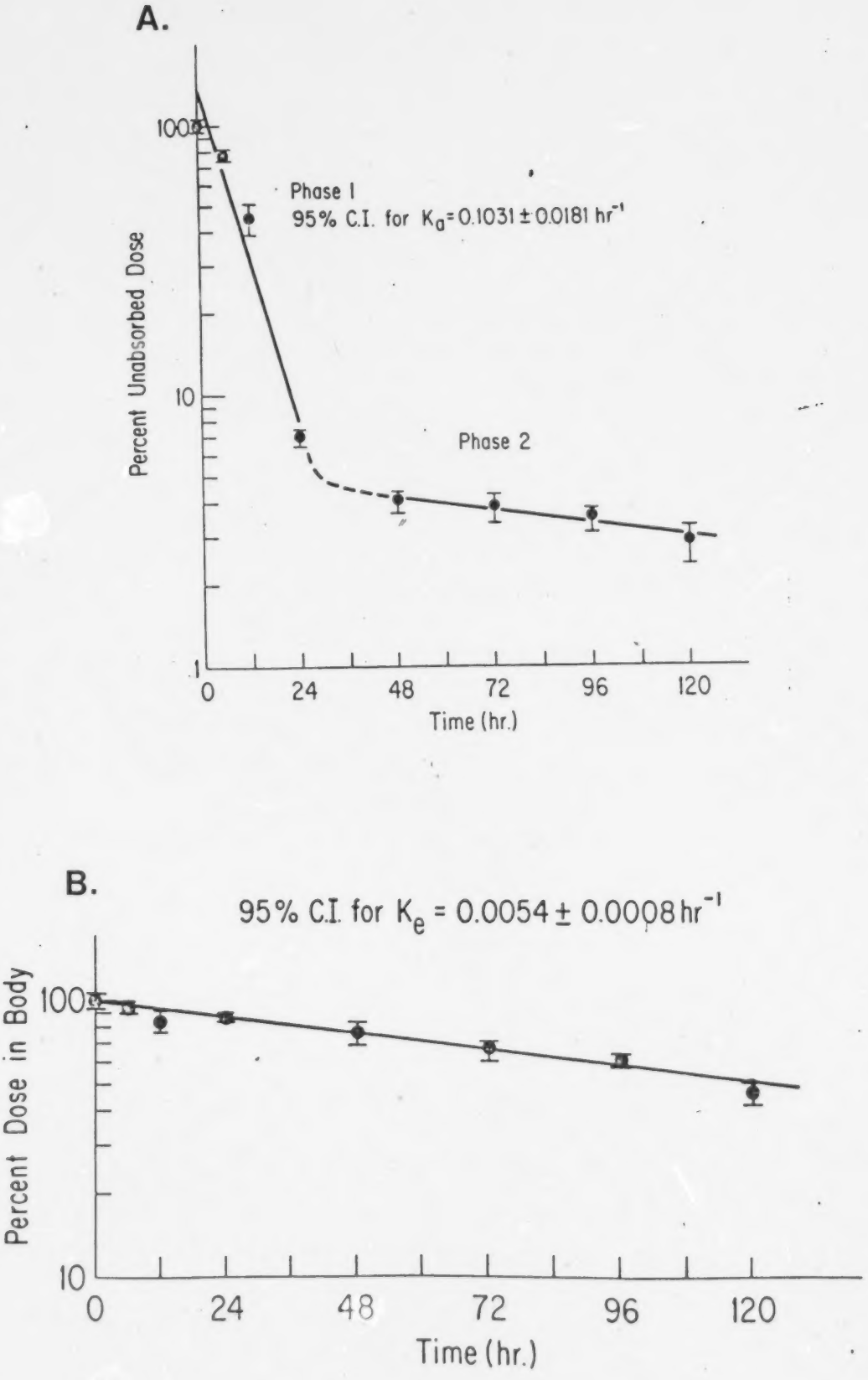

A.
100
10
1
Percent Unabsorbed Dose
Phase 1
95% C.I. for K_a = 0.1031 ± 0.0181 hr^{-1}
Phase 2
0
24
48
72
96
120
Time (hr.)
B.
95% C.I. for K_e = 0.0054 ± 0.0008 hr^{-1}
100
10
Percent Dose in Body
0
24
48
72
96
120
Time (hr.)

Fig. 9 The fractional absorption of a single dose by first-order absorption and elimination. The solid curve was determined as in Table 4 and the data of the current study where $k_e/k_a = 0.05$. A maximum of 85 percent was absorbed by 30 hr. The other curves are presented for comparison (Goldstein et al., 1974) at the same k_e. The case where $k_e/k_a = 0$ ($k_a = \infty$) is attained by constant input rate. For $k_e/k_a = 0.50$, where for a k_a smaller than that of the solid curve, a lower maximum would be attained at a later time.

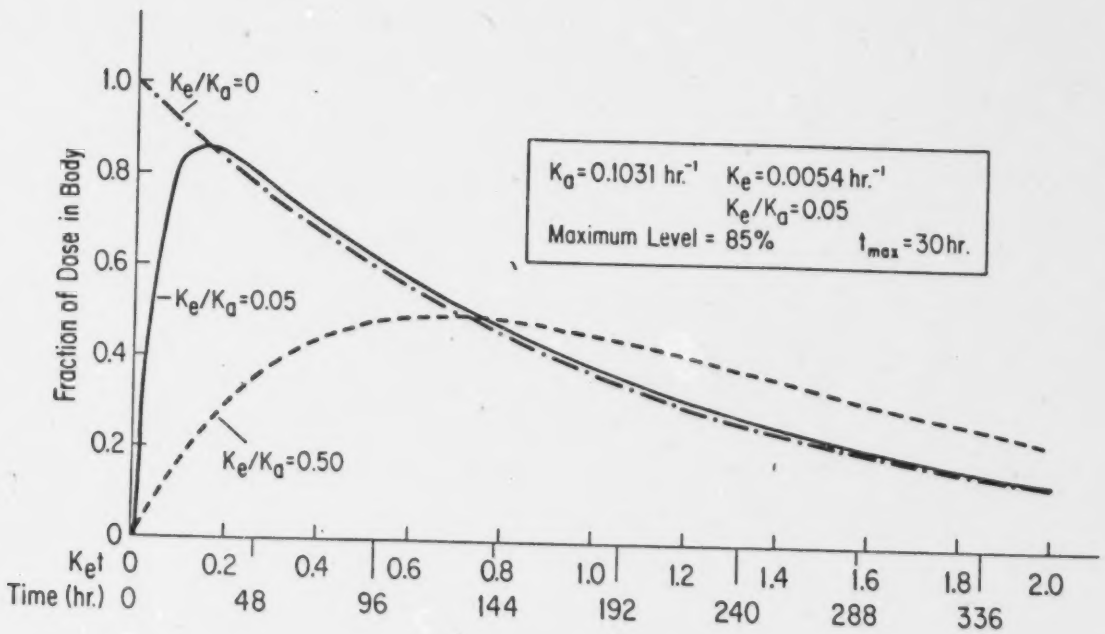
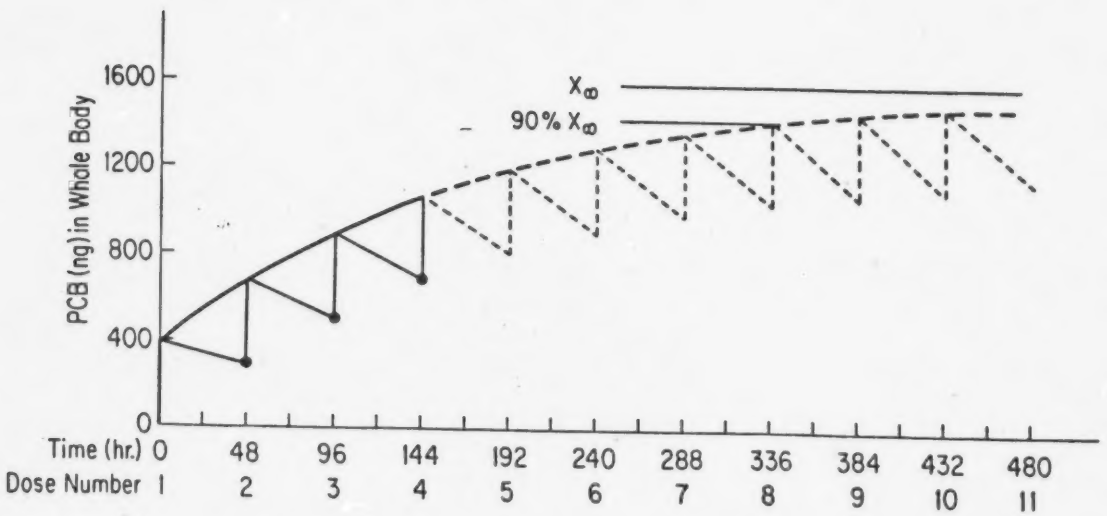

1.0
0.8
0.6
0.4
0.2
0
Fraction of Dose in Body
K_e/K_a=0
K_e/K_a=0.05
K_e/K_a=0.50
K_a=0.1031 hr.$^{-1}$ K_e=0.0054 hr.$^{-1}$
K_e/K_a=0.05
Maximum Level = 85% t_{max}=30hr.
K_et 0 0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8 2.0
Time (hr.) 0 48 96 144 192 240 288 336

Fig. 10 Curve for the cumulative retention of PCB from multiple dosing. Solid lines present actual levels attained during experiment. Dashed lines are the calculated extension of the data. The plateau burden (X_∞) is the steady state level attained from peak values after sufficient dosing (see text).


PCB (ng) in Whole Body
1600
1200
800
400
0
X_∞
90% X_∞
Time (hr.) 0 48 96 144 192 240 288 336 384 432 480
Dose Number 1 2 3 4 5 6 7 8 9 10 11

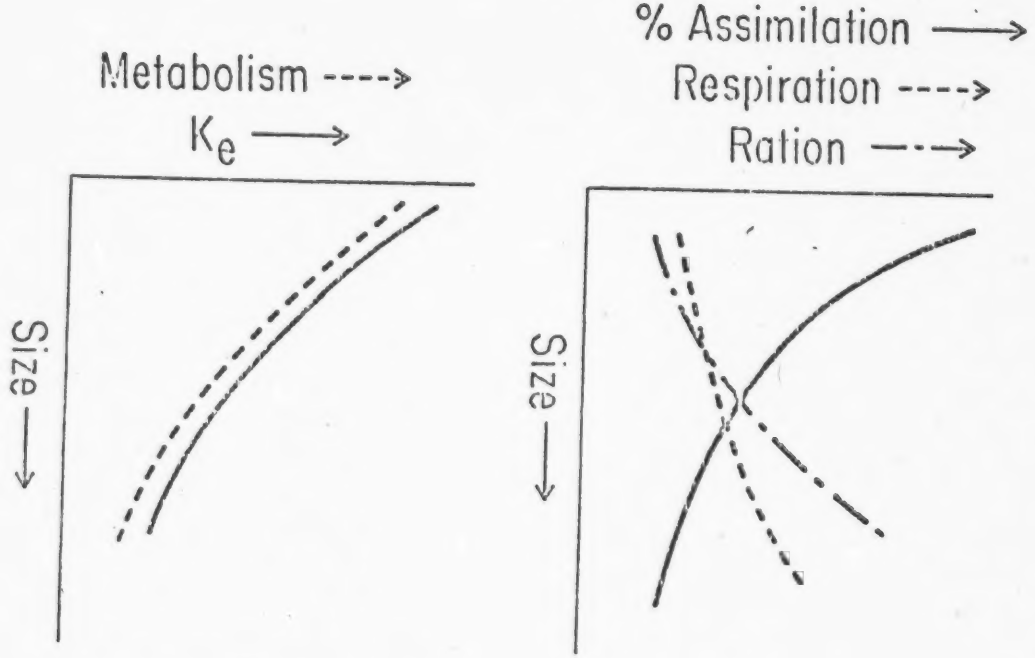
observe the approach to "steady-state". The feeding interval in nature is more likely to be twice each day. Mathematically, this is unimportant; what is essential is that the kinetics of the system (see TableVII) show plateau reached at dose n = 17, or after 32 days.

APPLICATION TO MODELING

Thomann (1981; see Fig. 7) documented clearly the complexity required of models describing contaminant accumulation in fishes. In his model he noted: 1) the lack of data relevant to the function referred to as the "food-chain multiplier"; and 2) the need to account for age-specific changes in respiration, feeding, and growth as determinants of predicted PCB body burdens in striped bass. In the preliminary model presented here we provide the food chain multiplier, as influenced by growth of the fish, changes in metabolism, changes in food ration, and changes in dietary PCB levels.

Pizza's analysis (1983) of striped bass PCB accumulation from diet revealed that changes in plateau PCB burden depended upon two factors. The first is the k_e. The second is the level at which new PCB is taken in via the food. The relationship between physiological growth and required ration size, as well as weight-specific metabolism, is depicted schematically in Fig. 11, along with the expected effects that changes in these factors may have on food

Fig. 11. Schematic representation of the response of various physiological parameters to increased size of the organism. In the upper figure, required ration to grow and respiration (µg O_2 per hour per individual) increase with increased size; assimilation efficiency of food decreases. In the lower figure, metabolism and elimination constant k_e decrease with increasing size.


Metabolism ---->
K_e ⟶
Size ⟵
% Assimilation ⟶
Respiration ---->
Ration —·→
Size ⟵

assimilation efficiency and the elimination constant (k_e) for PCBs (Califano et al., 1982; Pizza and O'Connor, 1983). The physiological data suggest that, for a growing fish, there should be no steady-state PCB level; since metabolism (and, hence, k_e) declines with age, and since assimilation efficiency declines with increasing age and ration required for growth increases, the PCB body burden should increase continously. Further, since the volume of water required for oxygen exchange must increase with size, more direct water uptake of PCB is possible. The importance of the latter in contributing to body burden may be questioned, however, since respiratory requirements may be extremely variable depending upon ambient levels of dissolved oxygen, temperature, time of day, time in the feeding cycle, etc. (Neumann et al., 1982).

The pharmacokinetic model incorporated these relationships under conditions representative of striped bass biology in the Hudson estuary. These were:

1. Active feeding during the first growing season on zooplankton containing 1 μg/g (dry weight) PCB (O'Connor et al., 1982).

2. An instantaneous growth rate of 0.02 d^{-1} (O'Connor and Bath, unpublished).

3. Decreasing k_e at a rate proportional to weight; $W^{-0.3}$ (Brett, 1981).

4. Increased ration size to maintain a daily intake of 10% body weight.

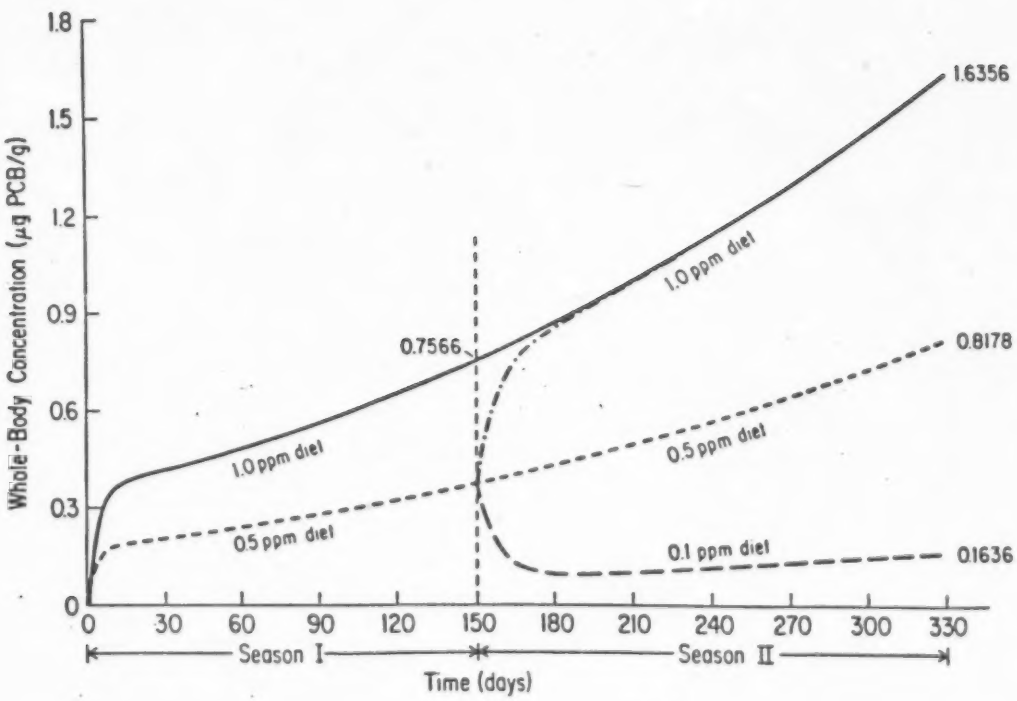
5. A feeding interval of 12 hr, with one-half the daily ration (and PCB dose) consumed during each feeding period.

6. Additional feeding on zooplankton containing either 0.5 µg/g, or 0.1 µg/g PCB (dry weight).

The model was run under several input conditions for two seasons of growth (total time 330 days). This exercise illustrates the temporal relationship of PCB input rate and output rate-constant within the boundaries of a realistic time scale (Fig. 12). Since the concentration in the diet was 1.0 ppm, the solid curve shows the computed BAF. The initial rise in concentration (0-30 days) was the predicted plateau-shift. Once plateau was attained, the typical asymptote did not occur, but rather the BAF skewed upward as determined by the rate of decline in k_e.

After one season of growth, the k_e declined to 0.0044 hr^{-1} and the computed BAF was 0.76. This value is much lower than the BAF = 0.95 which would be calculated by simply dividing the feeding rate by the k_e determined after 5 months of growth (0.0044

Fig. 12. Outcome of the pharmacokinetic model describing dietary PCB accumulation in striped bass. See text for details.


Whole-Body Concentration (μg PCB/g)
1.8
1.5
1.2
0.9
0.6
0.3
0
1.6356
1.0 ppm diet
0.7566
0.8178
1.0 ppm diet
0.5 ppm diet
0.5 ppm diet
0.1 ppm diet
0.1636
0 30 60 90 120 150 180 210 240 270 300 330
Season I
Season II
Time (days)

hr^{-1}). The discrepancy is explained by the fact that the BAF = 0.95 would be attained only with sufficient time (~ 44 days) and cessation of the decline in k_e, allowing for the establishment of a new steady state. This underscores the importance of recognizing that bioaccumulation is rarely characterized by steady state. but rather is a process of uninterrupted plateau shift operating as a continuum.

This point is illustrated by the dashed curves of Fig. 6. For a diet containing 0.5 ppm PCB, the time required for plateau-shift was the same as the 1.0 ppm situation, and the whole-body concentration was one-half the level of the higher dosage. Dividing the whole-body concentrations at any point in the curves by the PCB concentration in the respective diet shows that the BAF is the same for the two situations.

The more boldly dashed lines illustrated the expected outcome of a change in input rate during growth. If the PCB concentration in the diet is decreased, a plateau-shift will occur at a rate determined by the declining k_e. The whole-body concentration will decrease, but since k_e continues its decline with growth, the plateau will eventually skew upward maintaining the integrity of the BAF. This is demonstrated by dividing the whole-body concentration by that of the diet.

If the change is to a more concentrated diet, the whole-body

burden should reach a higher level. The model predicts that an increase to 1.0 ppm dietary PCB would cause a k_e-governed plateau-shift to the whole-body level, attained from a continuous 1.0 ppm dietary exposure (Fig. 12, solid curve), once again maintaining the BAF.

The growth rate in the model was determined by an assumed instantaneous rate-constant of 0.02 d^{-1}. This value was used because it is consistent with the growth rate of juvenile Hudson River striped bass, and results in approximately the same growth observed in the field.

The model was run for consecutive growth intervals (seasons I and II). Fluctuations in growth known to occur due to photoperiod, temperature, and other factors (Brett, 1979) were not considered. Such perturbations should elicit a response consistent with the k_e- and input-governed plateau shift.

A period of overwintering was not included in the calculations because relatively little is known of Hudson River striped bass physiological condition for this time of year. A reduction in feeding rate would be expected (Brett, 1979) along with a decline in metabolic rate (Neumann et al., 1981). The decline in body burden which is due to decreased input should more than offset the increased burden owed to depressed k_e. It is also important to realize that any reduction in k_e would cause an

increase in the time required for shifting plateau. Overall, a slow decline in body burden would be expected from overwintering unless utilization of stored lipid accelerates the reduction. Whatever level the body burden attains during periods of reduced metabolism, prediction based on growth would not change; once plateau-shift is complete, the PCB concentration in the fish should reflect the operant input rate and elimination rate constant.

When interpreted over a specific and realistic time-frame during life stages, the growth-related BAF model suggests that the PCB history of the fish may be inconsequential (disregarding all physiological perturbations) once a plateau-shift has occurred. Rather than simply interpreting field concentration data as the end result of prior PCB exposure, it would be more informative to use these data in bioaccumulation prediction as determined by life history with corresponding feeding and clearance rates.

Data are available for young-of-the-year striped bass taken in 1978 from the Indian Point portion of the Hudson River (Califano et al., 1982; Mehrle et al., 1982). Concentrations of PCB in these two samples were 1.59 and 2.62 μg/g (wet wt), or about 6.4 and 10.4 μg/g (dry wt), respectively. O'Connor's (1982) data for striped bass food organisms (Gammarus spp.) from the same portion of the Hudson averaged ~ 7 μg/g (dry wt) PCB. Given a BAF of 0.76

and a food source with 7 µg/g PCBs, one might expect a fish to contain 5.3 µg/g PCB (dry wt) due to diet alone.

This calculation suggests that 51 to 83% of the PCB in striped bass is due to dietary uptake. It is not implied that this might be the case for fishes in general; direct uptake from water is an important source of PCB to fishes, and cannot be ignored. However, the exercise does suggest that the dietary PCB component may be of great significance to fishes, especially in heavily contaminated environments such as the Hudson estuary.

As stressed throughout this discussion, the feeding rate is a critical factor in determining how much of the PCB burden was acquired via the diet. The model operates under two assumptions which concern the input rate and could affect the outcome of the predicted body burden. The first assumption is that the dietary PCB absorption efficiency is 100%. The current work with Aroclor 1254 showed that this level is not far from the efficiency observed, and should suffice since it also serves to simplify the model. For the limited data available, 51-83% would constitute a liberal assessment of the PCB burden obtained via the diet.

The second assumption involves the daily ration used in the model. A daily requirement of 10% body weight was chosen because this level of feeding is common to many studies (e.g., Chesney and Estevez, 1976; Phillips and Buhler, 1978). This level is

arbitrary, however, since an insufficient amount of work has been performed on the relation of physiological condition and growth rate to ration size. Compounding this problem is the fact that species, temperature, and body size affect the growth/ration relation. The few available studies usually deal with salmonoids at temperatures generally below 20°C (see Brett, 1979), and inference to the ration required by Hudson River striped bass is difficult. The sub-maximum growth rations reported by these studies are 8% or less. For the current work, where striped bass received an amount of food which filled the stomach without extension, a daily ration of 5% would have been feasible when based on two feedings a day. Laboratory striped bass of ~ 1 g (dry) can consume twice this quantity of live G. tigrinus when fed passively at 12 hr intervals (personal observation), and for the above reasons, a 10% daily ration was used in the model. Once again, before verification of the 51-83% dietary contribution is possible, an accurate value for ration is needed. It is also important to note that the BAF values (Fig. 6, solid curve) would be affected directly by the required reduction or increase in ration.

Dietary accumulation is most strongly influenced by feeding and clearance rates. The decline of these rates as growth occurs will determine the ultimate dietary contribution to body burden in mature fish. Certain environmental factors, such as reduced temperature, could serve to decrease k_e and increase "BAF" due to a

depressive effect on metabolic rates in striped bass and other fishes (Neumann et al., 1981). The impact of the reduced k_e under low temperature conditions, however, might be offset to some extent by simultaneous reduction in feeding during the winter months, or by metabolic compensation to slowly changing environmental conditions (Fry, 1971; Vetter, 1982).

The concept of growth-related plateau shift has application to estimating body burdens in fishes exposed to varying conditions of PCB input during different life-history stages. We know, for example, that when Hudson River striped bass migrate from riverine nursery areas to the lower estuary and marine waters, they show a significant reduction in PCB body burden (LMS, 1980; MacLeod et al., 1981; O'Connor et al., 1982). This reduction should be due to reduced PCB input, since the PCB in both water and food in coastal regions is less than in the estuary (Pierce et al., 1981; O'Connor et al., 1982). With knowledge of the operant k_e and the PCB levels in food and water, the body burden and time for reaching the new and lower level should be calculable. Research is currently underway to obtain data appropriate to making such predictions.

Norstrom et al. (1976) discuss the environmental and growth factors which influence contaminant accumulation through clearance rate. All these factors require study. When the data become

available, they can add significantly to the accuracy of predictive bioaccumulation models for PCB and other contaminants (e.g., Thomann, 1981; Mackay, 1982). The data from this study combined with observations from the field suggest strongly that dietary PCB sources are of great importance to striped bass. The combined effect of efficient cross-gut assimilation of PCB and low k_e dictates that PCB uptake from the food web be given additional attention in toxicological studies.

Our empirical studies and modeling data have direct application to ongoing ocean dumping studies as follows:

First, our hypothesis that PCB burden in fishes is strongly related to diet renders the "mass loading" approach to ocean PCB pollution ineffective in estimating contaminant levels in fish. Both dredged materials and sewage sludge must be evaluated more carefully to assess real quantities of PCBs injected into the food chain and the water column, rather than merely estimating mass loads placed in the ocean environment.

Second, the pharmacokinetics of PCBs in fishes suggests that any isolation of contaminated materials from entry to the food chain will have the effect of lowering PCB body burdens in the large, predatory species which often form an important part of the human diet. Thus, maintaining a surficial layer of sediments with low or non-available PCBs will, in all likelihood, result in a

trend toward lower body burdens in all trophic levels.

Third, the overall outcome of the model suggests that we shall not see increased PCB levels in fishes if input rates and input sources remain similar to those of recent years. Further dumping regulations should include attention to rendering such contaminants as PCB first, unavailable to food chains, and finally, unavailable in the water column.

VIII. FIELD TEST OF THE ECOKINETIC MODEL FOR PCB ACCUMULATION IN FISHES

INTRODUCTION

Previous sections in this report described laboratory studies designed to quantify the mechanisms of PCB accumulation in striped bass from water and from food sources. Our analyses of PCB kinetics and assimilation from the diet suggested that PCB-contaminated food may be the major source of body burdens in striped bass from the Hudson estuary (Pizza and O'Connor, 1983; Section VII). An accumulating body of evidence supports the thesis that chlorinated organics, and PCBs in particular, are accumulated in fishes more from dietary sources than by equilibrium partitioning from water (Mitchell et al., 1977; Califano, 1980; Thomann and Connolly, 1982; Pizza and O'Connor, 1983; Stehlik and Merriner, 1983; McKim and Heath, 1983).

If this is true for striped bass in a PCB-contaminated environment, and if the accumulation/elimination kinetics determined for PCBs in the laboratory are valid in nature, then field-verification studies should be possible (Pizza and O'Connor, 1983). The primary requirement would be that the subject population occupy a habitat for a time long enough to enable expression of shifts in "plateau" PCB levels. It would also be essential that the PCB environment be "stable", and that PCB burdens in food items or stomach contents be known.

This section presents the results of a field study conducted between August, 1982 and April, 1983. During this time samples of striped bass, stomach contents and food items were taken from various sites in the Hudson estuary and analyzed for PCB content. The fish col-

lected from January through March were overwintering in the New York Harbor region and, thus, represent a population limited in their movements to a relatively small area. The fish collected from August 1982 through January 1983 included young-of-year striped bass; sampling sites corresponded to nursery areas from August to October, 1982, and young-of-year fish taken in January were overwintering in the Harbor area (McLaren et al., 1981).

MATERIALS AND METHODS

Collections of striped bass were made from July, 1982 through April, 1983. The samples from July through October were taken using a 20 m (50 ft) seine with 64 mm mesh in the bag. Sampling sites were at Stony Point and Croton Point, N.Y., in the brackish water portion of the Hudson estuary used as a nursery ground by young-of-year striped bass (McLaren et al., 1981). The fish ranged in size from 28 mm to 57 mm (S.L.) during July, and from 66 to 86 mm (S.L.) in October. The collections were transported alive to the laboratory where representative samples were removed and sacrificed for analysis.

The winter samples (January through March) were taken in the New York Harbor region at Weehawken (January), Hoboken (March 2) and among the piers at Canal St. in Manhattan (29 March)(Fig. 2). The collections were made using a 12 m trawl with 1.3 mm stretch-mesh liner in the cod-end. The fish taken during the winter period comprised several age-classes, from 0+ (young-of-year) to 2+ (bass spawned in 1980). The striped bass taken in trawls were placed in plastic bags and held on ice in coolers for transport to the lab. They were sacrificed and processed for analysis within 24 hr of collection.

Each fish for analysis was weighed and the standard length was recorded. Stomachs were removed and the contents (if any) were examined to identify the food organisms to genus and species (where possible). Where stomach contents were of sufficient mass they were saved and frozen for PCB analysis. In some instances, stomachs were either empty, or the contents were too few for PCB analysis; in such cases the probable food items were inferred from other fish in the sample and the probable PCB content was inferred from the extensive data base we retain on PCB distribution on Hudson River zooplankton (O'Connor, 1982).

Fish up to 86 mm (S.L.) were subjected to whole-body PCB analysis. The fish were dried to constant weight at 50°C and ground in a mortar and pestle; about 1 g (when available) was used for PCB extraction. For larger fish (> 86 mm) a sample of epaxial muscle from the left side was removed, weighed, dried, and pulverized, and portion (1 g) was used for PCB extraction. Stomach contents and uningested food samples were dried, weighed and pulverized prior to extraction.

PCB extraction was carried out in acetonitrile (3X), using a Bransonic ultra-sound bath. The three extracts were combined and partitioned to n-hexane, followed by clean-up on florisil using 15% v/v ethyl ether/hexane as the eluent. Samples were reduced in volume under a N_2 stream and analyzed on a Varian Model 3700 GC with a ^{16}Ni ECD. The column was a fused-silica capillary column with SE-54 as the stationary phase. Quantitation of PCBs in individual peaks was performed by a Spectra-Physics Model 2000 integrator with parameters obtained from analysis of U.S. EPA standard PCBs (Aroclor 1254 and 1016). Isomer class identification was predicated upon analysis of selected chlorobi-

phenyl isomers (Cl_1 to Cl_6).

QA/QC data on PCB analyses was as follows: Recovery from spikes was 95%, n = 24; Procedure variation was ± 8.3% based on sample splits prior to extraction; Instrument variation was 9.5% based upon repeat analyses of single extracts, n = 12. Inter-lab comparison of unknowns with U.S. EPA Gulf Breeze (GBERL) and with N.Y. State Dept. of Health (DOH) labs yielded similar values at ± 14% (GBERL) and ± 20% (DOH).

RESULTS AND INTERPRETATION

1982 Year Class Striped Bass: Representatives of the 1982 year class of Hudson River striped bass were taken in samples from July, 1982 through January, 1983. During this time the year class was growing rapidly, increasing from a mean wet weight of 2.3 ± 0.8 (July 1982) to 20.7 ± 3.6 grams. This rate of growth approximates the average growth rate of 0.02 d^{-1} for Hudson River striped bass as measured by Dey et al. (1981). PCB concentrations in the 1982 year class fish were highest during July (10.8 ± 3.2 µg/g dry weight) and lowest in the January, 1983 sample (1.5 ± 0.5 µg/g dry).

The PCB concentrations decreased by a factor of 7.2 in the 160-day period between 28 July and 4 January, while the size of the fish sampled increased by a factor of 9. Total body burdens of PCBs in the July samples compared to those taken in January, 1983 showed an increase of about 32%. Thus, we conclude that while concentrations in the fish decreased, the overall burdens in the fish did not; the data should not be construed as evidence for simple depuration of PCB associated with time.

We demonstrated in an earlier section that the rate constant for PCB elimination (k_e) was equal to 0.005 hr^{-1}, based upon ^{14}C-Aroclor 1254 studies of whole-body PCB burdens. By applying the body burden approach to the present data it can be seen that there was, in fact, no net loss of PCB during the specified time interval. Rather, there exists a similar burden or a slight increase in burden with time. This suggests that either PCBs are not being eliminated in the environment, or that the rate of PCB accumulation between July and January is roughly equal to the rate of elimination. The latter of the two hypotheses is the more likely, since there exist measurable concentrations of PCBs in Hudson River water and in striped bass food organisms throughout the Hudson system (O'Connor, 1982; O'Connor et al., 1982; O'Connor and Pizza, 1983; Section VII, this report). These data may be used as a test for the ecokinetic model (Section VII), using as input data the PCB content of food at the upriver (Stony Point) station and the PCB content of food items at the downriver (Weehawken) station as well as estimated rates of growth, feeding rates, basic pharmacokinetics (Section VI) reduction in k_e associated with growth-related metabolic changes (Section VII) and migratory movement of the striped bass.

The results of model runs predict that, during the first 150 days of feeding, young-of-year striped bass would accumulate 7.6 µg/g PCB due to diet if food organisms were assumed to contain 10 µg/g PCBs. The value for PCB in food organisms is consistent with data collected for food organisms in the Stony Point region (O'Connor, 1982), and results in a food-related burden between 70 and 90% of the values observed at Stony Point and Croton Point in July, 1982. In fact, the predicted food-related body burdens are within one standard deviation of the

observed data.

Striped bass generally move to downriver locations for overwintering (McLaren et al., 1981). In January 1983, we measured PCB values in 1982 year class fish at 1.5 µg/g. These fish had available to them food organisms containing about 4 µg/g PCBs. According to the ecokinetic model, a change to food organisms containing lower levels of PCB would result in a downward plateau shift. The new plateau would be dependent upon PCB dose, k_e and the dosing interval. Given a food concentration of 4 µg/g and assimilation/elimination rates equivalent to those obtaining at upriver sites, the predicted duration of the plateau shift would be 25 days, with the minimum attained equal to 6.5 µg/g PCB derived from diet.

The predicted value of 6.5 µg/g must be modified, however, in order to account for several factors. These are: 1) reduced rate of feeding during overwintering; 2) reduced growth rates during winter; and 3) generally lower metabolic levels during the overwintering period. Using data from Neumann et al. (1981) which estimate the effect of temperature on striped bass metabolism, we calculated that overall metabolic activity of bass would decrease by a factor of approximately 3 concomitant with a 20°C reduction in temperature (from 25°C to 5°C). Assuming linearity in metabolic systems, this would have the overall effect of reducing both feeding rate and k_e by 3. In the model this would result in a lengthening of the time to plateau (from 25 to 75 days) and reducing the food intake by a factor of 3. The outcome is a predicted minimum body burden of 2.2 µg/g reached at 75 days (mid-January) for fishes ingesting food organisms at 4 µg/g at a reduced rate. The calcu-

lated burden is, in fact, quite similar to that observed for 1982 year class striped bass in the January sample.

These data suggest strongly that the ecokinetic model has a strong predictive capacity. However, many aspects of the model require refinement and confirmation in order to provide body burden estimates that are consistent with known physiological and behavioral parameters. Most critical among these are: 1) determining empirically that k_e declines with growth; 2) determining the effect of reduced temperature on k_e and feeding rate; 3) estimating the frequency of feeding during winter months; and 4) determining whether food conversion efficiency and the PCB assimilation rate constant (k_a) change with temperature.

Despite these required data, the field test of the model provided support for a variety of important features pointed up by the model. These are: 1) that PCB burdens in fishes are not fixed once accumulated, since elimination processes appear to go on in nature, even under contaminated conditions; 2) that estimates of the dietary component of PCB burdens in fish appear to be consistent at percentages generally above 75% of the total burden; and 3) that the time course for dietary PCB accumulation by fishes is a predictable quantity conforming to pharmacokinetic models. This last point further supports the importance of dietary uptake as the primary route for PCB accumulation, since the time to plateau via diet vastly exceeds that calculated for uptake via the water route.

REFERENCES

Armstrong, R.W. and R.J. Sloan. 1980. Trends in levels of several known chemical contaminants in fish from New York. N.Y. State Dept. Env. Conserv. Div. Fish Wildlife Tech. Rept. 80-2: 77 pp.

Bederka, Jr., J., A.E. Takemori and J.W. Miller. 1971. Absorption rates of various substances administered intramuscularly. Eur. J. Pharmacol. 15: 132-136.

Boehm, P.D. and P. Hirtzer. 1982. Gulf and Atlantic survey for selected organic pollutants in finfish. U.S. Dept. of Commerce, NOAA Tech. Mem. NMFS-F/NEC-13. 111 pp.

Bopp, R.F. H.J. Simpson, C.R. Olsen and N. Kostyk. 1981. Polyehlorinated Biphenyls in sediments of the tidal Hudson River, N.Y. Environ. Sci. Technol. 15:210-216.

Borgstrom, B. 1974. Fat digestion and absorption. IN: Biomembranes Vol. 4B (Manson, L.A. ed.). Plenum Press, New York, pp. 555-620.

Branson, D.R., G.E. Blau, H.C. Alexander and W.B. Neely. 1975. Bioconcentration of 2,2',4,4' tetrachlorobiphenyl in rainbow trout as measured by an accelerated test. Trans. Am. Fish Soc. 104: 785-792.

Brett, J.R. 1979. Environmental factors and growth. IN: Fish Physiology, Vol. 8; Bioenergetics and Growth (Hoars, W.S., D.J. Randall and J.R. Brett, eds.). Academic Press, New York, pp. 599-675.

Brown, M.P., J.J.A. McLaughlin, J.M. O'Connor and K. Wyman. 1982. A Mathematical model of PCB bioaccumulation in plankton. Ecological Modelling. 15: 29-47.

Brown, M.P., M.B. Werner and R.J. Sloan. 1985. In press. Recent trends in distribution of polychlorinated biphenyls in the Hudson River System. Environ. Sci. Technol.

Bruggemann, W.A., L.B.J.M. Marton, D. Kooiman and O. Hutzinger. 1981. Accummulation and elimination kinetics of di-, tri- and tetrachlorobiophenyls by goldfish after dietary and aqueous exposure. Chemosphere. 10: 811-832.

Cahn, P., J. Foehrenbach and W. Guggino. 1977. PCB levels in certain organs of some feral fish from New York State. IN: Physiological Responses of Marine Biota to Pollutants (Vernberg, J., A. Calabrese, F. Thurberg and W. Vernberg, eds.). Academic Press, Nre York, pp. 51-61.

Califano, R.J. 1981. Accumulation and tissue distribution of polychlorinated biphenyls (PCBs) in early life stages of the striped bass, Morone saxatilis. Ph.D. Diss. New York University. 163 pp.

Califano, R.J., J.M. O'Connor and J.A. Hernandez. 1982. PCB dynamics in Hudson River striped bass. I. Accumulation in early life history stages. Aquatic Toxicology 2: 187-204.

Califano, R.J., J.M. O'Connor and L.S. Peters. 1980. Uptake, retention and elimination of PCB (Aroclor 1254) by larval striped bass, Morone saxatilis. Bull. Env. Contam. Toxicol. 24(4): 467-472.

Cantilli, R.E.. 1979. The effects of polychlorinated biphenyls and acetoneon the respiration of Gammarus tigrinus Sexton 1939. M.S. Thesis Biology Dept. New York University, 44 pp.

Carcitch, I.G. and T.J. Tofflemire. 1982. Distribution and concentration of PCB in the Hudson River and associated management problems. Envirnoment Int. 7: 73-86.

Chesney, E.J. and J.I. Estevez. 1976. Energetics of winter flounder (Pseudopleuronectes americanus) fed the polychaete Nereis virens under experimental conditions. Trans. Amer. Fish Soc. 5: 592-595.

Clayton, J.R., S.P. Pavlou and N.F. Breitner. 1977. Polychlorinated biphenyls in coastal marine zooplankton: Bioaccumulation by equilibrium partitioning. Environ. Sci. Technol. 11: 676-681.

DiToro, D. and L.M. Horezempa. 1982. Reversible and resistant components of PCB adsorption-desorption: Isotherms. Environ. Sci. Technol. 16: 594-602.

Duke, T.W., J.I. Lowe and A.J. Wilson, Jr.. 1970. A polychlorinated biphenyl (Aroclor 1254) in the water, sediment and biota of Escambia Bay, Florida. Bull. Environm. Contam. Toxicol. 5: 172-180.

Fisher, N.S. 1975.. Chlorinated hydrocarbon pollutants and photosynthesis of marine phytoplankton: a reassessment. Science 189: 463-465.

Fry, F.E.J. 1971.. IN: The Effect of environmental factors on the physiology of fish, Vol. 6 (Hoar, W.S. and D.J. Randall eds.) Academic Press, New York, pp. 1-98.

Ginn, T.C.. 1977. An ecological investigation of Hudson River macrozooplankton in the vicintiy of a nuclear power plant. Ph.D. Diss. New York University, Dept. of Biology. 272 pp.

Goldstein, A., L. Arnow, and S.M. Kalman. 1974. Principles of Drug Action: The Basis of Pharmacology. J. Wiley and Sons, New York. 854 pp.

Gruger, E.H., Jr., N.L. Karrich, A.I. Davidson and T. Hruby. 1975. Accummulation of 3,4,3',4', tetrachlorobiphenyl and 2,4,5,4',5', and 2,4,6,2',4',6' hexachlorobiphenyl in juvenile coho salmon. Environ. Sci. Technol. 9: 121-127.

Guiney, P.D. and R.E. Peterson, 1980. Distribution and elimination of polychlorinated biphenyl after acute dietary exposure in yellow perch and rainbow trout. Arch. Environ. Contam. Toxicol. 9: 667-674.

Guiney, P.D., Peterson, M.J. Melancon, Jr. and J.J. Lech. 1977. The distribution and elimination 2,5,2',5', . ^{14}C- tetrachlorobiphenyl in rainbow trout. Toxicol. Appl. Pharmacol. 39: 329-338.

Hamelink, J.L., R.C. Waybrant and R.C. Ball. 1971. A proposal: exchange equilibria control the degree hydrocarbons are biologically magnified in lentic environments. Trans. Am. Fish Soc. 100: 207-221.

Hiraizumi, Y., M. Takahashi and M. Nishimura. 1979. Adsorption of polychlorinated biphenyl onto seabed sediment, marine plankton and other adsorbing agents. Env. Sci. Technol. 13: 580-584.

Hutizinger, O., D.M. Nash, S. DeFreitas, R. Norstrom, D. Wildish and V. Zitko. 1972. Polychlorinated biphenyl: Metobolic behavior of pure isomers in pigeons, rats and rainbow trout. Science 178: 312-314.

Hutzinger, O., S. Safe and V. Zitko. 1974. The Chemistry of PCBs. CRC Press, Cleveland, Ohio. 269 pp.

Interstate Electronics Corporation (IEC). 1981. Environmental Impact Statement for New York Dredged Material Disposal Site Designation. I.E.C., Anaheim, California.

Jensen, S. 1966. Report of an new chemical hazard. New Sci. 32: 612.

Karickhoff, S. 1984. Pollutant Sorption in Environmental Systems. Ch. 3. In: W.B. Neily and G.E. Blau (eds.). Environmental Exposure from Chemicals. Boca Raton, FL. CRC Press. In press.

Krzeminski, S.F., J.T. Gilbert and J.A. Ritts. 1977. A pharmacokentic model for predicting pesticide residues in fish. Arch. Environ. Contam. Toxicol. 5: 157-166.

Kuratsune, M., Y. Masuda and J. Nagayama. 1976. Some recent findings concerning Yusho. Proc. Nat'l. Conf. on PCBs EPA-560/6-75-004, pp. 14-30.

Lawler, Matusky and Skelly Engineers (LMS). 1980. Biological and water quality data collected in the Hudson River near the proposed Westway Project during 1979-1980. Rept. to N.Y. State Dept. of Transportation, Vol. II, LMS Engineers, Pearl River, New York.

Lee, G.F. and R.A. Jones. 1977. An assessment of the environmental significance and chemical contaminants present in dredged sediments dumped in the New York Bight. Rept. to U.S. Army Corps of Engineers, N.Y. District. 62 pp.

Lee, R.F. 1977. Accumulation and turnover of petroleum hydrocarbons in marine organisms. IN: Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems (Wolfe, D.A., ed.). Pergamon, New York, pp. 60-70.

Lieb, A.J., D.D. Bills and R.O. Sinhuber. 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (salmo gairdnerii). J. Agr. Food Chem. 22: 638-642.

Mackay, D. 1982. Correlation of bioconcentration factors. Environ. Sci. Technol. 16: 274-278.

MacLeod, W.D., Jr., L.S. Ramos, A. Friedman, D. Burrows, P. Prohaska, D. Fisher and D. Brown. 1982. Analysis of residual chlorinated hydrocarbons, aromatic hydrocarbons and related compounds in selected sources, sinks and biota of New York Bight. U.S. Dept of Commerce NOAA Tech. Memo. OMPA-6. 128 pp.

Mayer, F.L., P.M. Mehrle and H.O. Sanders. 1977. Residue dynamics and biological effects of polychlorinated biphenyl in aquatic organisms. Arch. Environ. Contam. Toxicol., 5: 501-511.

McKim, J.M. and E. M. Heath. 1983. Dose determinations form waterborne 2.5.2',5', (^{14}C) tetrachlorobiphenyl and related pharmacokenitic in two species of trout (Salmo gairdneri and Salvelinus fontinalis): A mass balance approach. Toxicol. Appl. Pharmacol. 108: 177-187.

McLeese, D.W., D.C. Metcalf and D.S., Pezzak. 1980. Bioaccumulation of chlorobiphenyl and endrin from food by lobsters (Homarus americanus). Bull. Environ. Contam. Toxicol. 25: 161-168.

Mehrle, P.A., T.A. Haines, S. Hamilton, L. Ludke, F.L. Mayer and M. Ribick. 1982. Relationship between body contaminants and bone development in east-coast striped bass. Trans. Am. Fish Soc. 111: 231-241.

Melancon, M.J. and J.J. Lech. 1976. Isolation and identification of a polar metabolite of tetrachlorobiphenyl form bile of rainbow trout exposed to ^{14}C-tetrachlorobiphenyl. Bull. Environm. Contam. Toxicol. 15: 181-188.

Mitchell, A.I., P.A. Plack and I.M. Thompson. 1977. Relative concentrations of ^{14}C-DDT and of two polychlorinated biphenyls in the lipids of cod tissue after a single oral dose. Arch. Environ. Contam. Toxicol. 6: 525-532.

Morales, N.M., D.B. Tuhey, W.A. Colburn and H.B. Matthews. 1979. Pharmacokinetics of multiple oral doses of selected polychlorinated biphenyls in mice. Toxicol. Appl Pharmacol. 48: 397-407.

Mueller, J.A., T.A. Gerrish and M.C. Casey. 1982. Contaminant inputs to the Hudson Raritan estuary. N.S. Dept. of Commerce, NOAA Tech. Memo. OMPA-21.

Nadeau, R.J. and R.A. Davis. 1976. Polychlorinated biphenyls in the Hudson River (Hudson Falls-Fort Edward) New York. Bull Environ. Contam. Toxicol. 16: 436-444.

Narbonne, F.J.. 1979. Accumulation of polychlorinated biphenyl (Phenclor DP6) by estuarine fish. Bull. Environ. Contam. Toxicol. 22: 60-64.

National Academy of Sciences-National Research Council. 1979. Polychlorinated biphenyl. National Academy of Sciences Press, Washington, D.C.

Nau-Ritter, G.M.. 1980. The dynamics of PCB transfers among marine phytoplankton, clay particles and water. M.S. Thesis Marine Sciences Research Center, State Univ. N.Y. at Stony Brook.

Neely, W.B., D. Branson and G. Blau. 1974. Partition coefficient to measure bioconcentration potential of organic chemicals in fish. Environ. Sci. Technol. 8: 1113-1115.

Nelson, N. 1972. PCBs-Environmental Impact. Env. Rsch. 5: 249-362.

Neumann, D.A., J.M. O'Connor and J.A. Sherk, Jr. 1981. Oxygen consumption of white perch (Morone americana) striped bass (M. saxatilis) and spot (Leiostomns xanthurus). Comp. Biochem. Physiol. (A) 69: 467-478.

Neumann, D.A., J.M. O'Connor, J.A. Sherk, Jr. and K.V. Wood. 1982. Respiratory response of striped bass (Morone saxatilis) to suspended solids. Estuaries 5: 44-55.

New York State Dept. Env. Cons. 1981. Toxic Substances in Fish and Wildlife. May 1 to November 1, 1981. Tech. Rept. Vol. 4, No. 2. TR82-1 (BEP). 45 pp.

New York State Dept. Env. Cons. 1982. Toxic Substances in Fish and Wildlife. November 1, 1981 to April 30, 1982. Tech. Rept. Vol. 5, No. 1. TR82-2 (BEP). 25 pp.

Norstrom, R.J., A.E. McKinnon and A.S.W. DeFreitas. 1976. A Bioenergetics-based model for pollutant accumulation by fish. Simulation of PCB and methylmercury residue levels in Ottowa River yellow perch (Perca flavescens). J. Fish Res. Bd. Canada 33: 248-267.

O'Brien and Gere Engineers, Inc. 1979. Bioaccumulation studies on Homarus americanus. Rept. to U.S. Army Engineers District, New York. 52 pp.

O'Connor, D.J. and J.P. Connolly. 1980. The effects of concentration of adsorbing solids on the partition coefficent. Water Research, 14: 1517-1523.

O'Connor, J.S. and H.M. Stanford (eds). 1979. Chemical Pollutants of the New York Bight; Priorites for Research. U.S. Dept. of Commerce, NOAA, ERL. 217 pp.

O'Connor, J.M. 1982. Biological Monitoring of PCBs in the Hudson River estuary. Rept. to N.Y. State Dept. of Environmental Conservation, Bureau of Water Research. 202 pp.

O'Connor, J.M. 1984, in press. Polychlorinated biphenyls in the diet of striped bass in New York Harbor. Northeastern Env. Sci., Vol. 3.

O'Connor, J.M., J.B. Klotz and Theo. J. Kneip. 1982. Sources, sinks and distribution of organic contaminants in the New York Bight esosystems. IN: Ecological Stress and the New York Bight (Mayer, G., ed.) Estuarine Res. Fed. Charleston, S.C., pp. 631-652.

O'Connor, J.M. and J.C. Pizza. 1984. Pharmacokinetic model for PCB accumulation in marine fish. IN: Wastes in the Ocean, Vol 9 (Duedall, I., ed.). Academic Press, N.Y. In press.

Pastel, M., B. Bush and J.S. Kim. 1980. Accumulation of polychlorinated biphenyls in American shad during their migration in the Hudson RIver, Spring, 1977. Pestic, Mon. Journal 14: 11-20.

Pavlou, S.P. and R.N. Dexter. 1979. Distribution of polychlorinated biphenyls (PCB) in estuarine ecosystems. Testing the concept of equilibrium partitioning in the marine environment. Environ. Sci. Technol. 13: 65-71.

Pequegnat, W.B., B.M. James, C.A. Kennedy, A.D. Fredericks, R.R. Fay and F. Hubbard. 1980. Application of the biotal ocean monitor system to a study of the impacts of ocean dumping of dredged material in the New York Bight. TerEco Rept. to U.S. Army Engineers, N.Y. District. 61 pp.

Peters, L.S. and J.M. O'Connor. 1982. Factors affecting short-term PCB and DDT accumulation by zooplankton and fish from the Hudson estuary. IN: Ecological Stress and The New York Bight (Mayer, G., ed.). Estuarine Research Federation, Charleston, S.C., pp. 451-467.

Peterson, R.E. and P.D. Guiney. 1979. Disposition of polychlorinated biphenyls in fish. IN: Pesticide and Xenobiotic Metabolism in Aquatic Organisms (Khan, M.A., J.J. Lech and J.J. Mann eds.). Amer. Chem. Soc., Wahington, D.C., ACS Symp. Series.

Phillips, G.R. and D.R. Buhler. 1978. The relative contributions of methyl-mercury from food and water to rainbow trout (Salmo gairdneri) in a controlled laboratory environment. Trans. Am. Fish Soc. 107: 853-861.

Pierce, R.J., T.D. Wright and J.M. O'Connor. 1981. Biomagnification: Relationship to dredged material disposal in the coastal marine environment. Estuaries 4: 258-299.

Pinkney, A.E. 1982. Uptake and retention of ^{14}C-Aroclor 1254 in Gammarus tigrinus after dietary exposure with the fungus Fusarium oxysporum. M.S. Thesis, N.Y. Univ., Dept. of Biology. 70 pp.

Pizza, J. 1983. Pharmacokinetics and distribution of dietary PCBs in Hudson River striped bass, Morone saxatilis. Ph.D. Thesis, N.Y. Univ., Dept. of Biology. 109 pp.

Pizza, J.C. and J.M. O'Connor. 1983. PCB dynamics in Hudson River striped bass. II. Accumulation from dietary sources. Aquatic Toxicol. 3: 313-327.

Risebrough, R.W., P. Reiche, D. Peakall, S. Herman and M.N. Kirven. 1968. Polychlorinated biphenyls in the global ecosystem. Nature 220: 1098-1102.

Rubinstein, N.I., E. Lores and N. Gregory. 1983. Accumulation of PCBs, mercury and cadmium by Nereis, virens, Mercenaria mercenaria and Palaemonetes pugio from contaminated harbor sediments. Aquatic Toxicol. 3: 249-260.

Rubinstein, N.I., W.T. Gillian and N. Gregory. In press. Dietary accumulation of PCBs from a contaminated sediment source by a demersal fish (Leiostomus xanthurus). Aquatic Toxicol.

Sloan, R.J. and R.W. Armstrong. 1982. PCB patterns in Hudson River fish. II. Migrant-marine species. Hudson River Env. Soc. Symposium 1982. Hudson River Env. Soc., New York, N.Y.

Spagneli, J.J. and L.C. Skinner. 1977. PCBs in fish from selected waters of New York State. Pestic. Monitor. Jour. 11: 69-87.

Stehlik, L. and J.V. Merriner. 1983. Effects of accumulated dietary kepone on spot (Leiostomus xanthurus). Aquatic Toxicol. 3: 345-358.

Tavolaro, J.F.. 1982. Sediment budget study for clamshell dredging and disposal activities. U.S. Army of Engineers New York District. 27 pp.

Thomann, R.V.. 1978. Size-dependent model of hazardous substances in aquatic food chains. EPA Report: EPA-600/3-78-036. 39 pp.

Thomann, R.V.. 1981. Equilibrium model of fate of microcontaminants in diverse aquatic food chains. Can. J. Fish Aquatic Sci. 38: 280-296.

Thomann, R.V. and J. St. John. 1979. The fate of PCBs in the Hudson River ecosystem. Ann. N.Y. Acad. Sci. 320: 610-629.

Thomann, R.V. and J.P. Connolly. 1984. An age dependent model of PCB in a Lake Michigan food chain. Env. Sci. Technol., 18: 65-71.

Turk, J.T.. 1980. Applications of Hudson River Basin PCB Transport studies. IN: Contaminants and Sediments, Vol. II (Baker, R.A. ed.). Ann Arbor Science Press, Ann Arbor, MI, pp. 171-185.

Turk, J.T. and D. Troutman. 1981. Polychlorinated biphenyl transport in the Hudson River, New York. U.S. Geological Survey Water Res. Invest. 81-9, U.S.G.S. Albany, N.Y. 11 pp.

U.S. Environmental Protection Agency (EPA). 1980. Ambient water quality criteria for polychlorinated biphenyls. EPA 440/5-80-068, U.S. EPA, Washington, D.C. 191 pp.

Vetter, R.D.. 1982. Seasonal metabolic compensation in sympatric seatrout: Adaptation to the estuary. Trans. Am. Fish. Soc. 111: 193-198.

Wasserman, M., D. Wasserman, S. Cucos and H. Miller. 1979. World PCBs Map: storage and effects in man and his biologic environment in the 1970's. Ann. N.Y. Acad. Sci. 320: 69-124.

Weininger, D. 1978. Accumulation of PCBs by lake trout in Lake Michigan. Ph.D. Diss. Univ. Wisconsin Madison. 232 pp.

West, R.H. and P.G. Hatcher. 1980. Polychlorinated biphenyls in sewage sludge and sediments of the New York Bight. Mar. Poll. Bull. 11: 126-129.

Wildish, D.J., C.D. Metcalf, H.M. Akagi and D.W. McLeese. 1980. Flux of Aroclor 1254 between estuarine sediments and water. Bull. Environ. Contam. Toxicol. 24: 20-26.

World Health Organization (WHO). 1975. Environmental health criteria for polychlorinated biphenyls (PCBs) and terphenyls (PCTs). WHO environmental Health Criteria Program. 97 pp.

Wyman, K. and H.B. O'Connor. 1980. Implications of short-term PCB uptake by small estuarine copepods (genus Acartia) from PCB contaminated water. Est. Coatal Mar. Sci. 11: 121-131.

Zar, J.H. 1974. Biostatistical Analysis. Prentice-Hall, Englewood Cliffs, N.J. 620 pp.